IN THE UNITED STATES DISTRICT COURT
FOR THE SOUTHERN DISTRICT OF WEST VIRGINIA
AT BECKLEY

RYAN HYSELL and CRYSTAL HYSELL,
on behalf of their daughter, A.H., a minor,

Plaintiffs,

v. Civil Action No. 5:18-cv-01375

RALEIGH GENERAL HOSPITAL,
and THE UNITED STATES OF AMERICA,

Defendants.

PROPOSED FINDINGS OF FACT AND CONCLUSIONS OF LAW OF THE UNITED STATES OF AMERICA

I. PROPOSED FINDINGS OF FACT

1. On October 29, 2010, Crystal Hysell was admitted to Raleigh General Hospital ("RGH") for the labor and delivery of her first child. (JM_1029.)[1] Ms. Hysell was at 41 weeks of gestation. (JM_1072; JM_2018.) Ms. Hysell was admitted to RGH at 04:53. (JM_1029.)

2. The fetal heart rate tracing on admission was normal with a normal baseline, normal variability, and the absence of any significant decelerations. (JM_3001–JM_3002; Trial Tr. at 1904:8-17 (Landon Test.).)[2] At 07:30, the cervix was four centimeters dilated and completely effaced with the vertex at minus two station. (JM_1030.) Just after 08:30, an epidural was placed. (JM_1031.) Labor progressed normally such that at 12:22, Ms. Hysell was noted to be dilated to

[1] Citations to "JM" reference Bates numbers located on Defendants' Joint Defense Exhibits 1 through 3. In the record, Joint Defense Exhibit 1 consists of JM_1001–JM_1192, (ECF No. 284); Joint Defense Exhibit 2 consists of JM_2001–JM_2114, (ECF No. 284-1); and Joint Defense Exhibit 3 consists of JM_3001–JM_3061, (ECF No. 284-2).

[2] Citations to the trial transcript are to its consecutively paginated numbers. The transcript, as divided into volumes, appears on the record as follows: Volume I consists of pages 1–247, (ECF No. 293); Volume II consists of pages 248–504, (ECF No. 294); Volume III consists of pages 505–706, (ECF No. 295); Volume IV consists of pages 707–955, (ECF No. 296); Volume V consists of pages 956–1222, (ECF No. 297); Volume VI consists of pages 1223–1419, (ECF No. 298); Volume VII consists of pages 1420–1641, (ECF No. 299); Volume VIII consists of pages 1642–1823, (ECF No. 300); Volume IX consists of pages 1824–2007, (ECF No. 301); and Volume X consists of pages 2008–2146, (ECF No. 302).

nine centimeters, (JM_1035), she began to feel felt rectal pressure at 12:25, and she was fully dilated at 12:50 and pushing, (JM_1036). At 13:10, the fetal tracing reveals a mildly elevated baseline of 170 beats per minute ("bpm"). (JM_3046–JM_3047.) No significant decelerations were noted, and variability was normal. (Trial Tr. at 1907:1–23 (Landon Test.).) At 13:50, the tracing was entirely reassuring. (JM_3051; Trial Tr. at 1909:19–1910:1 (Landon Test.).) At 14:00, Ms. Hysell was instructed to push. (JM_1037.) At 14:01, the tracing revealed a baseline elevated to 180 bpm. (JM_3052.)

3. Debra Crowder, CNM, a former labor and delivery nurse of almost ten years and certified nurse midwife of over fifteen years at Access Health, (Trial Tr. at 168:9, 185:9-15 (Crowder Test.)), attended to Ms. Hysell. At 11:40, CNM Crowder noted that Ms. Hysell was feeling pressure (status-post epidural), a vaginal examination was 8/100/0, spontaneous rupture of the membranes revealed clear fluid, the fetal heart rate was in the 150s with accelerations and moderate variability, uterine contractions were every two to four minutes, and labor was progressing. (JM_1081.) She recommended a position change to the left side, and she anticipated a single vaginal delivery. (JM_1081.)

4. CNM Crowder's note at 14:10 indicated Ms. Hysell was pushing with good effort, the fetal heart rate was in the 140s to 150s with variables up to 60 to 80 when pushing and moderate variability along with uterine contractions every two minutes. (JM_1081.) Her plan was for Ms. Hysell to continue to push, and she anticipated a single vaginal delivery. (JM_1081.)

5. Ms. Hysell's labor progressed normally with no indication of any fetal distress or problems according to CNM Crowder and the RGH nursing staff. (JM_2018; Trial Tr. at 199:8-12 (Crowder Test.).) The tracing remained one of a well-oxygenated fetus. (Trial Tr. at 199:8-12 (Crowder Test.); Trial Tr. at 1916:5-16 (Landon Test.).) At 14:19, the chart notes that Labor and

Delivery Nurse Perkowski placed internal fetal scalp electrodes to monitor the fetal heart rate, replacing the external fetal heart monitor which had been used up until that time. (JM_1037.) At 14:30, the tracing was entirely reassuring and remained so for the duration of the labor and delivery. (JM_3056–JM_3059; Trial Tr. at 1911:21–1914:11 (Landon Test.).)

6. CNM Crowder does not document every time she goes in and out of a patient's room—to do so would be overly burdensome and unnecessary, and there is no evidence that RGH or Access Health's policies required it. While the record in this case does not reflect how long before A.H.'s delivery CNM Crowder arrived in the room, (Trial Tr. at 175:3-15 (Crowder Test.)), it is CNM Crowder's practice to visit each patient's room periodically even before delivery begins. The record in this case reflects that CNM Crowder was present in Ms. Hysell's room at least three different times prior to delivery at 11:40, 14:10, and 14:51. (JM_1039; JM_1081.)

7. As Plaintiffs' midwife expert, Mr. John Fassett, agreed,[3] Ms. Hysell experienced a normal second stage of labor, (Trial Tr. at 357:21–358:6 (Fassett Test.)), and she delivered a female child ("A.H.") through a spontaneous vaginal delivery at 14:55. (JM_1038; JM_1081; JM_2018.) The child weighed six pounds thirteen ounces at birth. (JM_1038; JM_2018.) Debbie Crowder, CNM, handed A.H. to the RGH nursing staff after the delivery and had no further contact with A.H. (Trial Tr. at 177:25–178:2 (Crowder Test.); Trial Tr. at 348:17-25 (Fassett Test.).) Mr. Fassett had no opinions or criticisms regarding the standard of care during Ms. Hysell's prenatal care or after the delivery of A.H. (Trial Tr. at 345:12-20, 348:6-16 (Fassett Test.).)

[3] Mr. Fassett was the sole standard of care witness called by Plaintiffs with regard to CNM Crowder. None of the of the other experts called by Plaintiffs expressed standard of care opinions regarding CNM Crowder. (Trial Tr. at 406:19–407:5 (Connors Test.); Trial Tr. at 566:14-18, 662:15-21 (O'Meara Test.); Trial Tr. at 688:20–689:3 (Rugino Test.); Trial Tr. at 807:17-24 (Lampton Test.); Trial Tr. at 874:16-18 (Staller Test.); Trial Tr. at 1184:24–1185:1 (Barakos Test.).)

8. A.H. had Apgar scores of seven at one minute and eight at five minutes, (JM_1073; JM_2018), with acrocyanosis (some bluish discoloration on her extremities caused by post-birth circulatory system development), (JM_2032). An Apgar score of seven or eight is considered normal and is a reassuring sign that the newborn was born in good health. (Trial Tr. at 1551:15–1552:12 (Giannone Test.); Trial Tr. at 1840:12-17 (Bedrick Test.).) A score of ten is very unusual since almost all newborns lose at least one point for blue hands and feet (acrocyanosis), which is normal after birth and not a finding indicating a medical problem. (Trial Tr. at 201:21–202:2 (Crowder Test.); Trial Tr. at 391:18-20 (Fassett Test.); Trial Tr. at 629:23–630:1 (O'Meara Test.); Trial Tr. at 1562:10 (Giannone Test.); Trial Tr. at 1842:2-8 (Bedrick Test.).)

9. The electronic fetal monitoring strip did not indicate any fetal distress or unusual problems during the progression of Ms. Hysell's labor. (JM_3001–JM_3061; Trial Tr. at 195:17-19 (Crowder Test.); Trial Tr. at 1916:5-16 (Landon Test.).) The electronic fetal monitoring strip had moderate variability throughout. (JM_3001–JM_3061; Trial Tr. at 195:20-23 (Crowder Test.); Trial Tr. at 1917:14–1918:6 (Landon Test.).) Moderate variability, like the presence of accelerations, indicates good fetal oxygenation. (Trial Tr. at 194:7-8, 195:24–196:1, 200:2-3 (Crowder Test.); Trial Tr. at 1902:1-22 (Landon Test.).) Thus, the electronic fetal monitoring strip pattern was not consistent with hypoxia. (Trial Tr. at 194:9-13, 195:17-22, 196:21-24 (Crowder Test.); Trial Tr. at 1917:14–1918:6 (Landon Test.).) On the contrary, "the fetal heart rate monitoring remained reassuring with good variability . . . throughout the entire strip until the baby was delivered." (Trial Tr. at 199:8-12 (Crowder Test.). *See also* Trial Tr. at 372:13-19 (Fassett Test.) ("Q. But in terms of variability, any time you could see the fetal heart rate there was variability, was there not? A. At that moment, correct. Q. Correct. So you never saw an absent – absence of variability on this strip, did you? A. I don't believe so, no.").)

10. RGH nursery personnel were present at delivery and took over the management of the child after delivery. (Trial Tr. at 177:25–178:2 (Crowder Test.).) After cleaning, suctioning, and warming, A.H. was transported by the RGH nursery staff to the nursery. (JM_2033; Trial Tr. at 1611:18–1612:25 (Ball Test.).) Again, CNM Crowder's role in caring for Ms. Hysell and A.H. was complete once delivery occurred. (Trial Tr. at 177:25–178:2 (Crowder Test.); Trial Tr. at 348:17-25 (Fassett Test.).)

11. A.H. was evaluated in the nursery at 15:08 by nurse Heather Buchanan. (JM_2032.) A.H. appeared to be acrocyanotic and a little dusky, and her oxygen saturation was measured at 68 percent, which "simply represents her individual, unique transition to extrauterine life." (Trial Tr. at 1842:15–1843:25 (Bedrick Test.).) Nurse Buchanan suctioned A.H. with a bulb syringe, removed a small amount of thick clear mucus, and applied blow-by oxygen. (JM_2032; Trial Tr. at 220:9–221:8, 222:24–223:7 (Buchanan Test.).) A.H.'s oxygen saturation rate then rose into the mid to high 90s range, which is normal for a baby. (JM_2033; Trial Tr. at 1854:7-10 (Bedrick Test.).) The nursing staff then suctioned the child with a catheter and removed a moderate amount of thick clear mucus. (JM_2033.) A.H. was weaned from the blow-by oxygen after ten minutes and continued to have a normal oxygen saturation rate. (JM_2033.) A.H. had no other respiratory problems during the remainder of her course in the nursery prior to discharge from the hospital.

12. Prior to birth, a baby in the mother's uterus or womb receives nutrients and oxygen through the placenta, which means that the baby's lungs do not function and the baby does not have normal blood flow—the baby is not breathing air. (Trial Tr. at 1834:14-23 (Bedrick Test.); Trial Tr. at 1553:23–1554:3 (Giannone Test.).) Once the baby is born and the umbilical cord is cut, an "amazing" and "miraculous" transition occurs, resulting in changes in the baby's

physiology of the heart and lungs that allows blood to flow to the lungs and the lungs' air sacs to progressively begin to expand with air. (Trial Tr. at 1834:24–1835:9 (Bedrick Test.); Trial Tr. at 1553:13–1554:3 (Giannone Test.).) Importantly, this transition to extrauterine life can vary greatly from baby to baby. (Trial Tr. at 1741:10-17 (Bedrick Test.); Trial Tr. at 1525:17-18 (Ball Test.).) There are many changes happening with the baby's physiology, and while some babies can make the transition seamlessly, other babies require a longer period of time for blood to flow adequately into the lungs, allowing the lungs to begin receiving oxygen from the atmosphere. (Trial Tr. at 1835:10-22 (Bedrick Test.); Trial Tr. at 1554:4-13 (Giannone Test.).)

13. The usual oxygen saturation rate of a baby in utero is between 50 and 60 percent.[4] (Trial Tr. at 1835:23–1836:4 (Bedrick Test.); Trial Tr. at 1553:5-12 (Giannone Test.).) While it generally takes a number of minutes for the oxygen saturation rate to begin increasing through the transition to extrauterine life, it is expected that babies around ten or fifteen minutes old will have saturations in the high 80s or low 90s. (Trial Tr. at 1836:5-10 (Bedrick Test.).) However, because the transition varies between babies, "the remarkable piece about this process is that not every baby will do it that quickly and some simply take a longer period of time." (Trial Tr. at 1836:10-13 (Bedrick Test.).) Thus, A.H.'s oxygen saturation reading of 68 percent less than fifteen minutes after birth simply represents A.H.'s "individual, unique transition to extrauterine life." (Trial Tr. at 1842:25–1843:3 (Bedrick Test.); Trial Tr. at 1587:7–1588:11 (Giannone Test.); Trial Tr. at

[4] Plaintiffs' pediatric expert, Dr. Alia Marie Iqbal O'Meara, did not know this number but recognized that a fetus's normal oxygen saturation level in utero is "low." (Trial Tr. at 637:2-8 (O'Meara Test.).) Plaintiffs' labor and delivery nursing expert on the other hand, Ms. Patricia Connors, believed it to be "90 to 100 percent" and was "surprise[d]" when she was told that the level (according to publications by the American College of Gynecologists and Obstetricians and the American Academy of Pediatricians) is between 50 and 60 percent in utero. (Trial Tr. at 432:22–433:10 (Connors Test.).) When asked whether someone who purports to be an expert in newborn nursing should "know that the Sa02 for a child in utero is not 95 to 100 percent," Dr. O'Meara conceded that such a person should know better. (Trial Tr. at 637:16-20 (O'Meara Test.).) When asked what the normal SaO_2 for a child in the womb was, RGH's Labor and Delivery Nurse (who attended to A.H.) as well as both neonatology experts answered at trial, without hesitation, that it is 50 to 60 percent. (Trial Tr. at 231:8-11 (Buchanan Test.); Trial Tr. at 1553:5-12 (Giannone Test.); Trial Tr. at 1835:23–1836:4 (Bedrick Test.).)

1944:6-17 (Landon Test.).) Her clinical presentation and swift increase and stabilization in oxygen saturation to the mid to high 90s range after deep suctioning and the application of blow-by oxygen, which is normal protocol for babies, make clear that the "brief observation of a low oxygen saturation played no role in causing any brain injury." (Trial Tr. at 1843:4-25 (Bedrick Test.); Trial Tr. at 1556:22–1557:9 (Giannone Test.).)

14. The nursery staff performed a physical examination of A.H., which was essentially normal except in one respect. A.H.'s head circumference was measured to be 31.9 centimeters. (JM_2032.) This measurement indicated that the child was born with microcephaly, (Trial Tr. at 1074:15-18 (Rugino Test.); Trial Tr. at 1263:3-11 (Shimony Test.); Trial Tr. at 1543:2-18 (Giannone Test.); Trial Tr. at 1803:2-8 (Scher Test.); Trial Tr. at 1846:24–1847:4 (Bedrick Test.)), a term used to describe a severely small head and usually defined as a head circumference less than two standard deviations below the mean or below the third or fifth percentile, (Trial Tr. at 1864:23–1865:10 (Bedrick Test.)). Dr. Rugino, an expert called by Plaintiffs, likewise agreed that A.H. was born with microcephaly. He testified that her head circumference was at the first percentile. (Trial Tr. at 1058:9-21; 1074:15-18 (Rugino Test.).) Because microcephaly takes many weeks or months to develop as it relates to brain growth, microcephaly at birth cannot be caused by anything that occurs close to the delivery process; whatever caused it must have occurred "[m]uch earlier in pregnancy, . . . [p]robably somewhere in the first-third or the beginning of the second-third" trimester. (Trial Tr. at 1847:5-25 (Bedrick Test.).)

15. A.H.'s course after birth in the nursery was otherwise normal, her pediatrician found no problems, and she was released two days post-delivery after a normal hospital stay. (JM_2008; JM_2043–JM_2046.) Her acrocyanosis also resolved in a normal fashion. (Trial Tr. at 1842:11-14 (Bedrick Test.).) She did not have any seizures, organ failure, or other medical

issues prior to her discharge from the nursery and the hospital. (Trial Tr. at 1845:22–1846:8 (Bedrick Test.).) A.H. did not show any signs of neonatal encephalopathy, neurological injuries, or acute neurologic syndrome in the nursery. She had a normal course in the nursery prior to her discharge from RGH. (Trial Tr. at 645:24–646:1 (O'Meara Test.); Trial Tr. at 774:22–775:18 (Gropman Test.); Trial Tr. at 1795:2–1796:19, 1797:10–1800:7 (Scher Test.); Trial Tr. at 1839:19–1840:25, 1842:15–1843:16, 1844:1–1845:2 (Bedrick Test.).)

16. During the first couple years of A.H.'s life, her parents reported to various medical providers that both Ms. Hysell's labor and A.H.'s delivery were normal and that there were no problems or complications. (ECF No. 286-3 at 1 (RGH Ex. 117); ECF No. 286-4 at 2 (RGH Ex. 118); ECF No. 286-5 at 1 (RGH Ex. 119).)

17. At approximately six months of age, A.H. appeared to begin missing some developmental milestones. (ECF No. 281-14 at 1 (Pls. Ex. 32c); ECF No. 281-25 at 2–5 (Pls. Ex. 11); Trial Tr. at 452:5-11 (Arthur Test.).) An MRI performed at Charleston Area Medical Center ("CAMC") in 2012 was interpreted as normal, (ECF No. 281-26 at 27 (Pls. Ex. 12)), and early genetic testing did not reveal any genetic anomalies to explain A.H.'s developmental delay, (ECF No. 281-23 (Pls. Ex. 7); Trial Tr. at 486:22–487:10 (Schorry Test.)).

18. A.H.'s parents took her to Cincinnati Children's Hospital for additional testing. A.H. was found to still be microcephalic by the physicians at Cincinnati Children's Hospital. (ECF No. 281-25 at 1 (Pls. Ex. 11); Trial Tr. at 453:5-6, 454:20, 461:2-10 (Arthur Test.) ("She had a small head size, microcephalic. . . . Microcephalic. Definitely. Her head was small."); Trial Tr. at 487:13-17 (Schorry Test.) ("Q. And what did you find on your physical exam? . . . A. . . . Her head circumference was about the third percentile.").) A further genetic work-up did not detect any genetic anomalies, (Trial Tr. at 488:6-13, 489:4-9, 493:21-25 (Schorry Test.)), and A.H. was

eventually diagnosed as having Autism Spectrum Disorder ("ASD"), (ECF No. 281-25 at 1 (Pls. Ex. 11)).

19. In 2016, the physicians at Cincinnati Children's Hospital decided to have another MRI performed of A.H.'s brain after she continued to miss some developmental milestones. The neuroradiologist at Cincinnati Children's Hospital interpreted that MRI scan as showing periventricular white matter anomalies—more commonly referred to as periventricular leukomalacia ("PVL"). (ECF No. 281-7 at 1 (Pls. Ex. 14); ECF No. 281-13 (Pls. Ex. 32B).) That neuroradiologist reviewed the MRI scan performed at CAMC in 2012 and determined that MRI scan had not been accurately interpreted. (ECF No. 281-14 at 2 (Pls. Ex. 32c); Trial Tr. at 459:11-13 (Arthur Test.) ("So . . . I basically humbly disagreed with the original reading when she was 18 months of age. I disagreed with it being a normal MRI.").) The neuroradiologist concluded that the 2012 MRI scan showed essentially the same findings discovered in the 2016 MRI scan. (ECF No. 281-14 at 2 (Pls. Ex. 32c); Trial Tr. at 459:5-11 (Arthur Test.).) The United States' expert in neuroradiology agreed with that assessment. (Trial Tr. at 1730:13-17 (Sze Test.).)

20. Microcephaly at birth suggests a prenatal insult early in pregnancy or abnormal development of the brain. (Trial Tr. at 1847:9-18 (Bedrick Test.); Trial Tr. at 1803:2-6 (Scher Test.).) Microcephaly at birth is not caused by an acute episode occurring at or near the time of birth. (Trial Tr. at 927:15-17 (Graham Test.); Trial Tr. at 1263:3–1264:7 (Shimony Test.) ("You can't cause microcephaly in acute injury at the time of birth.").) Rather, medical studies have indicated that microcephaly at birth may result from a prenatal insult early in pregnancy or abnormal brain development of the brain prior to birth. (Trial Tr. at 773:21–774:15 (Gropman Test.); Trial Tr. at 968:24–969:11 (Trock Test.) ("So a small head at birth indicates whatever may have happened to this poor girl's brain happened before birth – well before birth."); Trial Tr. at

1547:13–1548:8 (Giannone Test.); Trial Tr. at 1803:2-8 (Scher Test.) ("That suggests that some process happened prenatally and then you have to figure out when did that really happen and what trimester."); Trial Tr. at 1847:5-18 (Bedrick Test.) ("And in this circumstance, microcephaly is a process which takes many weeks or months to develop. It's nothing that occurred anywhere close to the delivery process. And so it reflects a process that for one reason or another did not allow her brain to grow as it should have.").)

21. In addition, PVL is a particular type of white matter disorder caused by an infectious or inflammatory hypoxic-ischemic event which frequently occurs between approximately 24 and 34 weeks of gestation. (Trial Tr. at 972:24–973:5 (Trock Test.); Trial Tr. at 1257:13-21, 1326:4-14 (Shimony Test.); Trial Tr. at 1743:22–1744:2 (Sze Test.).) Some children who experience PVL are born prematurely while others are born at term. (Trial Tr. at 1738:25–1741:22 (Sze Test.).) The injury is not caused by hypoxia at birth. (Trial Tr. at 1745:2-6 (Sze Test.).) The effects of PVL may not manifest themselves until many months after birth, but PVL can cause cerebral palsy, developmental delays, cognitive disorders, motor problems, and other types of neurological deficits. (Trial Tr. at 1279:14–1280:19 (Shimony Test.).)

22. A.H. has developmental disorders, cognitive delay, cerebral palsy (motor issues), and related deficits. (ECF No. 286-7 (RGH Ex. 121).) Her physicians continue to consider her to have ASD. (*See, e.g.*, ECF No. 286-6 (RGH Ex. 123). *But see* ECF No. 186-7 at 5 (RGH Ex. 121) ("AUTISM? Dr[.] Dodge thinks she has autism but parents just think she was really into her electronics when there. Her therapists have not thought she had autism. She does have that diagnosis from the past in Ohio, [her parents] cancelled the eval[uation] scheduled for December pending finalization of legal case.").)

23. The United States called several expert witnesses to testify at trial, including Dr. Mark Landon, Chairman of Obstetrics and Gynecology ("OB/GYN") at The Ohio State University School of Medicine, who is board-certified in OB/GYN and maternal-fetal medicine. (Trial Tr. at 1891:2-4, 1892:7-8 (Landon Test.).) Dr. Landon is one of roughly 100 out of 40,000 board-certified OB/GYNs in the United States to be selected as an examiner for the American Board of OB/GYN for a period of 25 years and has been chosen to do the same for board candidates in maternal-fetal medicine. (Trial Tr. at 1893:5-23 (Landon Test.).) Dr. Landon joined the faculty at The Ohio State University School of Medicine in 1987 and has held the titles of Assistant Professor, Associate Professor, and Full Professor in addition to Chairman of OB/GYN. (Trial Tr. at 1890:24–1891:4 (Landon Test.).) In addition to his administrative duties as Chairman, Dr. Landon still regularly delivers babies (as recently as the week before testifying at trial), attends and oversees the labor and delivery unit, and sees patients in an outpatient clinical setting. (Trial Tr. at 1891:11–1892:1 (Landon Test.).) Dr. Landon has written over 200 peer-reviewed articles and over 80 book chapters in the fields of obstetrics and maternal-fetal medicine, and, among other positions, he is Chief Editor for *Gabbe's Obstetrics*, now in its eighth edition. (Trial Tr. at 1894:8-18, 1895:12-17 (Landon Test.).) Dr. Landon was also a consultant and contributor for the American College of Obstetricians and Gynecologists and the American Academy of Pediatrics' *Joint Task Force Report on Neonatal Encephalopathy and Neurologic Outcome* (2d ed.) ("2014 Task Force Report").[5] (Trial Tr. at 1895:21–1897:2 (Landon Test.).) Finally, the majority of Dr. Landon's expert deposition testimony has been on behalf of plaintiffs, and about half of his trial testimony has been on behalf of plaintiffs. (Trial Tr. at 1922:12-23 (Landon Test.).) In fact, Dr.

[5] Dr. Landon is the only expert witness to testify at the trial of this case who was a consultant for this publication. Plaintiffs' pediatric expert, Dr. O'Meara, admitted that she has never read this publication, which is widely relied upon by physicians around the world who practice specialties where neonatal encephalopathy occurs. (Trial Tr. at 614:9-19 (O'Meara Test.).)

Landon has testified on behalf of a plaintiff against the United States in a case where the United States' counsel here was counsel of record. (Trial Tr. at 1922:24–1923:7 (Landon Test.).)

24. Dr. Landon has supervised and taught midwives as part of his practice for over fifteen years (including at the time of the events involved in this case) and was familiar with the standards of care that were applicable to midwives at the time of the events involved in this case. (Trial Tr. at 1897:3-22 (Landon Test.).) Throughout his career, he has reviewed tens of thousands of electronic fetal monitoring strips, including strips where children experienced hypoxic-ischemic insults during labor and delivery. (Trial Tr. at 1899:10-22 (Landon Test.).) He has taught medical students, residents, fellows, and midwives how to properly interpret strips for several decades. (Trial Tr. at 1897:25–1898:10 (Landon Test.).) Dr. Landon was familiar with the standards of care applicable to interpreting electronic fetal monitoring strips at the time of the events involved in this case. (Trial Tr. at 1898:11-15 (Landon Test.).)

25. Dr. Landon testified to the following opinions at trial to a reasonable degree of medical probability:

A. Based on the conduct of the labor, the fetal monitoring strips, and the actions associated with fetal monitoring, Debra Crowder, CNM, met acceptable standards of care for the management of Crystal Hysell's labor and delivery of her infant A.H. (Trial Tr. at 1915:2-15, 1924:3-14 (Landon Test.).) CNM Crowder still met the standard of care even if the testimony of Cindy Remines and Ryan Hysell regarding the presence of a cord and CNM Crowder's handling of it was true. (Trial Tr. at 1915:16–1916:4 (Landon Test.).)

B. Careful review of the fetal tracings fails to reveal changes consistent with any degree of in utero hypoxia capable of causing brain injury to the fetus. (Trial Tr. at 1916:5-16 (Landon Test.).) Dr. Landon has ***never*** seen a case where a baby has suffered a hypoxic-ischemic brain injury during labor and delivery yet had an electronic fetal monitoring strip showing normal variability and accelerations throughout like in this case. He also testified that he has ***never*** seen a baby have a hypoxic injury that occurred during labor and delivery based on the types of patterns seen in the electronic fetal monitoring strip involved in this case. (Trial Tr. at 1903:23–1904:3, 1917:19–1918:6; 1921:16-25 (Landon Test.).)

C. Dr. Landon reviewed the fetal monitoring strips in this case and concluded that until at least 12:22 the strip was a Category I strip, which is one that indicates no significant hypoxia and is entirely consistent with a metabolically normal fetus. (Trial Tr. at 1904:8-17 (Landon Test.).) While Plaintiffs emphasize testimony by their midwife expert, Mr. Fassett, that he interpreted a deceleration on panel 77112 of the fetal monitoring strip that represents "[p]ossibly likely cord compression of some sort," this panel reflects the time period of 12:10 to 12:11—a time during which Mr. Fassett had no criticisms of the care provided by CNM Crowder. (Trial Tr. at 311:22–312:15, 345:21–346:4 (Fassett Test.).) Moreover, as Dr. Landon testified, decelerations alone are not an indication of hypoxia. (Trial Tr. at 1912:19-21 (Landon Test.).)

D. As to the period of monitoring that Plaintiffs' midwife expert characterized as "uninterpretable" from approximately 12:22 to 14:20, (Trial Tr. at 343:13-16 (Fassett Test.)), Dr. Landon demonstrated to the Court that during that period, there was normal variability and accelerations and no late or repetitive decelerations, (Trial Tr. at 1905:11–1911:19 (Landon Test.)). Dr. Landon noted that the strip, even during this time, showed that A.H. was "not suffering from any hypoxia and certainly suggests that the fetus ha[d]n't suffered from any hypoxia capable of causing injury to the fetus." (Trial Tr. at 1907:3-7, 1916:25–1917:10 (Landon Test.).)

E. Dr. Landon highlighted two accelerations on the strip occurring less than ten minutes before birth, which represented that "the fetus [could] not be in any degree of significant hypoxia and certainly not acidotic or anything capable of being associated with hypoxic brain injury." (Trial Tr. at 1911:16-19, 1913:7-13 (Landon Test.).) Because there was no evidence that the child suffered from hypoxia during labor and delivery, any assertion that the tracing required different management, including earlier delivery, is simply false and without basis.[6] (Trial Tr. at 1911:4-8 (Landon Test.).)

F. Dr. Landon strongly disagreed with experts Dr. O'Meara's, Mr. Fassett's, and Ms. Connors' description of fetal tracings including the suggestion that multiple late decelerations were present in this case as such interpretations are not reasonable. (Trial Tr. at 1918:7-10 (Landon Test.).) He also disagreed with any suggestions that the tracing showing decelerations, lack

[6] As CNM Crowder testified, if she felt like she needed a second opinion based on the fetal monitoring strip, she would have brought in the OB/GYN. (Trial Tr. at 174:23–175:2 (Crowder Test.).)

of variability, and signs of fetal distress, using the strip for illustration. (*See, e.g.*, Trial Tr. at 1916:14-16, 1917:14–1918:6 (Landon Test.).)

G. The Apgar scores of seven and eight and immediate neonatal condition of A.H. were inconsistent with an acute intrapartum hypoxic injury to the fetus. (Trial Tr. at 1919:1–1920:13 (Landon Test.).) Further, the 2014 Task Force Report for which Dr. Landon was a consultant and contributor indicates that the labor and delivery in this case is not consistent with an acute hypoxic-ischemic event because the fetal monitoring strip was Category II, at worst, and A.H. had an Apgar score greater than seven at five minutes. (Trial Tr. at 1919:1–1920:13 (Landon Test.).)

H. If any hypoxic brain injury occurred to the fetus, it would have antedated the labor admission—"during the pregnancy itself." (Trial Tr. at 1920:14–1921:13, 1958:20–1959:4 (Landon Test.).)

I. The labor events and electronic fetal monitoring tracings do not support any degree of fetal hypoxia capable of causing hypoxic brain injury to A.H. during the labor and delivery process. (Trial Tr. at 1916:5-16, 1917:14–1918:18 (Landon Test.).) Given the tens of thousands of fetal monitoring strips that Dr. Landon has reviewed over the course of his career, he has ***never*** "seen a case, such as this with this pattern, where a baby was born with a hypoxic injury due to events which occurred during labor and delivery based upon this type of strip." (Trial Tr. at 1921:16-25 (Landon Test.).) Notwithstanding Plaintiffs' counsel's attempt to coax Dr. Landon into changing his opinion about the quality of the fetal monitoring strip, Dr.

Landon remained consistent that the strip does not show evidence of hypoxia during labor and delivery. (Trial Tr. at 1960:11-16 (Landon Test.).)

J. In sum, Dr. Landon opined that A.H.'s condition at birth, the Apgar scores at one and five minutes, and the patterns seen on the electronic fetal monitoring strip are inconsistent with an intrapartum hypoxic-ischemic injury to A.H. He concluded, to a reasonable degree of medical probability, that the child's brain injury predated Ms. Hysell's admission to labor and delivery. (Trial Tr. at 1921:4-9 (Landon Test.).)

26. The Court also heard testimony from Dr. Alan Bedrick, a board-certified pediatrician and neonatologist and Professor of Pediatrics and Emeritus Division Chief of Neonatology and Developmental Biology at the University of Arizona College of Medicine. (Trial Tr. at 1828:10-12, 1829:17-23, 1830:15-19 (Bedrick Test.).) Dr. Bedrick retains clinical responsibilities as an attending neonatologist at Banner University Medical Center in Tucson, Arizona, where he attends or cares for patients in the neonatal intensive care unit. (Trial Tr. at 1829:24–1830:5 (Bedrick Test.).) Dr. Bedrick has also served in the capacity of a neonatal intensive care unit medical director in various hospitals since the 1980s. (Trial Tr. at 1829:5-20, 1832:17-19 (Bedrick Test.).) In addition, he cares for babies who are transferred to neonatal intensive care because they are suspected of having experienced hypoxia during labor and delivery, including in cases that resulted in brain injuries. (Trial Tr. at 1832:12-16, 1837:24–1838:1 (Bedrick Test.).)

27. Dr. Bedrick testified to the following opinions at trial to a reasonable degree of medical probability:

A. A.H.'s current neurologic and developmental status is unrelated to any events in the perinatal labor and delivery and neonatal nursery time frames. (Trial Tr. at 1839:24–1845:2 (Bedrick Test.).)

B. A.H. did not suffer an acute hypoxic-ischemic brain injury in the labor and delivery time frame during her mother's admission to RGH, nor during the infant's neonatal hospital stay. (Trial Tr. at 1844:1-14 (Bedrick Test.).) She also showed no signs of acute neurologic syndrome during that same timeframe. (Trial Tr. at 1844:17–1845:2 (Bedrick Test.).)

C. A.H.'s Apgar scores were not consistent with a brain injurious event during the labor time frame prior to delivery. (Trial Tr. at 1840:12-17 (Bedrick Test.).) The infant had brief resuscitative efforts, which is not consistent with an acute event before delivery. (Trial Tr. at 1838:14-18 (Bedrick Test.).) In addition, the neurologic aspects of the physical examinations noted at multiple times during the neonatal hospital stay—notably the normal feeding behavior, activity, and tone—were not consistent with an acute hypoxic-ischemic brain injurious event during the labor and delivery or nursery time frame. (Trial Tr. at 1838:8–1839:6 (Bedrick Test.).)

D. It was critically important to note that the infant's multiple documented physical observations in the nursery at RGH were not consistent with a perinatal brain injurious process. (Trial Tr. at 1846:6-8, 1858:2-6 (Bedrick Test.).) Infants with such a brain injury in this time frame often require intubation and mechanical ventilation and have persistent and prolonged

hypotonia, stupor, depression, or coma. (Trial Tr. at 1838:8–1848:6 (Bedrick Test.).)

E. Dr. Bedrick concluded that baby A.H.'s physical examination and early clinical course strongly argue against a perinatal/neonatal etiology for her brain injury. (Trial Tr. at 1839:24–1840:9, 1846:6-8 (Bedrick Test.).) There was no clinical evidence of an acute neonatal encephalopathy immediately after delivery. (Trial Tr. at 1840:11-25 (Bedrick Test.).) More likely than not, A.H. did not sustain a hypoxic-ischemic event during labor and delivery, which resulted in brain injury. (Trial Tr. at 1839:24–1840:9 (Bedrick Test.).)

F. There was absolutely no clinical evidence of acute neonatal neurologic syndrome or evidence of an encephalopathic clinical picture in the immediate time frame post-delivery. (Trial Tr. at 1844:9-14 (Bedrick Test.). *See also* Trial Tr. at 645:24–464:1 (O'Meara Test.) ("Q. This child did not have neonatal encephalopathy on [October] 29th, 30th or 31st, correct? A. Correct.").) Such an observation is a clear requirement to make an associative link between a brain injurious process during labor and delivery or early neonatal period and subsequent permanent neurologic injury. (Trial Tr. at 1840:1-6, 1844:9-14, 1858:12-17 (Bedrick Test.).)

G. Babies who subsequently develop developmental delay or chronic neurologic impairment following an acute hypoxic-ischemic process in the perinatal/neonatal time frame are not discharged from the hospital in 48 hours, nor do they have a totally normal newborn nursery course and a

normal newborn neurologic physical examination at a discharge time of 48 hours. (Trial Tr. at 1839:7-18, 1859:24–1860:7 (Bedrick Test.).)

H. It was also significant to note that the infant was born with microcephaly with a head circumference falling less than the 3rd percentile. (Trial Tr. at 1846:24–1847:4 (Bedrick Test.).) This represents a profound disturbance of brain growth in utero, long pre-dating the labor and delivery and newborn time frame—probably occurring somewhere in the first trimester or the beginning of the second trimester. (Trial Tr. at 1847:9-25 (Bedrick Test.).)

I. Dr. Bedrick also did not agree with the opinions of Dr. O'Meara, Plaintiffs' pediatric expert, that A.H.'s subsequent neurologic difficulties were due to an acute perinatal hypoxic-ischemic event. (*Compare* Trial Tr. at 581:7-12, 582:17–583:4 (O'Meara Test.), *with* Trial Tr. at 1885:19–1886:1 (Bedrick Test.).) Dr. Bedrick testified that given the total absence of any neurologic symptoms or neurologic abnormalities in the newborn course, one cannot make any causal association between perinatal events and subsequent neurodevelopmental impairment. (Trial Tr. at 1882:17–1883:11 (Bedrick Test.).) Notably, Dr. O'Meara is not a neonatologist and does not work in a neonatal intensive care unit or nursery. (Trial Tr. at 600:19–601:12, 610:17–611:13 (O'Meara Test.).)

J. In sum, A.H.'s Apgar scores of seven and eight at one and five minutes of age, respectively, the lack of need for intubation or positive pressure ventilation, her initial and subsequent normal neurologic documentation, and her physical examinations did not indicate an operative brain injurious

process during labor and delivery or as a neonate. (Trial Tr. at 1834:4–1848:4 (Bedrick Test.).)

28. Dr. Mark Scher, a board-certified pediatric neurologist and neurophysiologist "with a particular interest in maternal and pediatric health dealing with fetal and neonatal neurology" also testified at trial. (Trial Tr. at 1776:4-6, 1781:19–1784:1 (Scher Test.).) Dr. Scher has over 36 years of experience as a clinician, educator, and researcher, (Trial Tr. at 1777:10-19 (Scher Test.)), and he is presently Emeritus Tenured Professor of Pediatrics and Neurology at Case Western Reserve University, (Trial Tr. at 1781:9-13 (Scher Test.)). Dr. Scher began that position on January 1, 2020, when he retired as Full Professor of Pediatrics and Neurology at Case Western Reserve University, but he continues to regularly mentor, teach, and write. (Trial Tr. at 1781:9–1782:3 (Scher Test.).) Dr. Scher served as Division Chief of Pediatric Neurology from 1997-2017. (Trial Tr. at 1780:6-13 (Scher Test.).) He has extensive clinical and research expertise in fetal and neonatal neurology, establishing and directing two fetal/neonatal neurology programs at the University of Pittsburgh, Magee-Women's Hospital from 1983-1997, and at Rainbow Babies and Children's Hospital/MacDonald Hospital for Women at University Hospitals Cleveland Medical Center from 1997 to the present time. (Trial Tr. at 1778:13-23, 1779:6–1781:2 (Scher Test.).) Dr. Scher has also published approximately 170 peer-reviewed articles and 44 book chapters in the field of pediatric neurology, and he serves on the editorial board and as a reviewer for several peer-reviewed journals. (Trial Tr. at 1784:14–1786:18 (Scher Test.).) Dr. Scher has testified for both plaintiffs and defendants, including at least one time for a plaintiff who sued the United States. (Trial Tr. at 1812:1-7, 1816:23–1817:7 (Scher Test.).)

29. Dr. Scher testified to the following opinions to a reasonable degree of medical probability at trial:

A. A.H. has a neurological condition comprised of a "constellation of multiple developmental domain delays having to do primarily with a social adaptive behavioral disorder called autism spectrum disorder." (Trial Tr. at 1789:1-4 (Scher Test.).) Her condition progressed on a developmental basis during fetal life—most likely beginning during the first trimester—with no relationship to the events at or around the time of delivery as a full-term infant. (Trial Tr. at 1802:5–1804:1, 1815:3-6 (Scher Test.).) A.H. was born with no evidence of significant neurological depression nor did she later express evidence of neonatal encephalopathy with or without seizures during the first several days of life. (Trial Tr. at 1796:1-19, 1816:8-19 (Scher Test.).) A.H. lacked clinical signs, laboratory abnormalities, and later brain MRI findings to support a diagnosis of intrapartum hypoxic-ischemic encephalopathy at full-term gestational age as discussed in a multidisciplinary consensus report. (Trial Tr. at 1796:20–1797:9, 1815:7-20 (Scher Test.).)

B. A.H. was microcephalic on her first postnatal day of life as her head circumference fell more than two standard deviations below the mean with all subsequent measurements "substantially in the second percentile." (Trial Tr. at 1803:2-5, 1813:18–1814:1 (Scher Test.).) Congenital microcephaly noted at birth strongly supports the diagnosis of significant delay in fetal head growth that more likely than not began during the first half of the mother's pregnancy. (Trial Tr. at 1802:6-11 (Scher Test.).)

C. A.H. subsequently exhibited a severe neurodevelopmental disorder across all four developmental domains of motor, communication, social-adaptive, and cognitive abilities, highlighting specifically abnormal language and social developmental abnormalities supporting the diagnosis of ASD. (Trial Tr. at 1789:1-6, 1790:23–1791:14, 1808:10-15 (Scher Test.).) Up to the present time, she continues to display this specific neurodevelopmental disorder. (Trial Tr. at 1789:1-6, 1806:18–1807:6 (Scher Test.).) This repertoire of abnormal behaviors is only later expressed during early childhood as the child's brain connections continue to mature, occurring because of a prenatal abnormality in brain development early in pregnancy of the mother. (Trial Tr. at 1808:10–1809:3 (Scher Test.).)

D. A.H.'s two brain MRI scans in 2012 and 2016 documented reduced brain volume with altered white matter signals consistent with this early developmental disorder of fetal brain maturation. (Trial Tr. at 1792:3-22 (Scher Test.).) These findings otherwise reflect a developmental disorder of the brain beginning "during the first half of pregnancy." (Trial Tr. at 1792:23–1793:2 (Scher Test.).) There was a preferential loss of brain substance in the posterior head regions resulting in disproportionate enlargement of the occipital horns of the lateral ventricles, i.e., colpocephaly. (Trial Tr. at 1792:18-22 (Scher Test.).) While these findings could be an early sign of asphyxia, i.e., hypoxic-ischemic injury, in a preterm brain, there is no indication of a clinically adverse event affecting mother's pregnancy. (Trial Tr. at 1793:20–1794:5 (Scher Test.).)

E. Alternatively, the neuroimaging finding could be a marker of a developmental anomaly affecting brain maturation due to a genetic disorder as early as at the time of conception. (Trial Tr. at 1793:4-8 (Scher Test.).) A.H. has a genetic disorder related to the MTHFR gene, (ECF No. 286 (RGH Ex. 122)), which affects the body's ability to metabolize a chemical called folate immediately after conception within the brain cells, (Trial Tr. at 1793:8-15 (Scher Test.)). This inability to metabolize during the brain's growth in utero eventually affects every cell of the brain and leads to developmental problems regarding language, thinking, behavior, and motor ability. (Trial Tr. at 1793:15-19 (Scher Test.).) Thus, the presence of the genetic abnormality "is very, very provocative and very helpful" in explaining A.H.'s neurological problems. (Trial Tr. at 1794:10-14, 1800:8–1801:1 (Scher Test.).)

F. In sum, A.H.'s ASD behaviors are unrelated to any acquired injury based on hypoxia-ischemia at any time during labor or delivery, and there were no clinical signs, laboratory testing results, or neuroimaging findings that support hypoxic-ischemic encephalopathy from an event or events that occurred during labor and delivery. (Trial Tr. at 1795:4-25, 1807:7-12, 1814:15–1816:22 (Scher Test.).)

30. Finally, the Court heard testimony from board-certified neuroradiologist Dr. Gordon Sze, a Harvard College and Harvard Medical School graduate who has taught at Yale University School of Medicine as a Professor of Radiology for the past twenty-four years and served as Chief of Neuroradiology for the past thirty. (Trial Tr. at 1723:4-24 (Sze Test.).) Dr. Sze

is a member of all the major scientific societies in his profession and was past-President of the American Society of Neuroradiology, the largest scientific society of radiology in the world. (Trial Tr. at 1724:19-25 (Sze Test.).) Dr. Sze was recently awarded the American Society of Spine Radiology's Gold Medal, which he described as an Oscar in his field based on past achievements. (Trial Tr. at 1725:1-7 (Sze Test.).) He has served on the editorial boards or as a reviewer for all the major radiology journals in his field, including the Journal of Radiology, American Journal of Neuroradiology (the largest scientific journal of neuroradiology in the world), Radiographics, Journal of Magnetic Resonance Imaging, Stroke, and many others. (Trial Tr. at 1725:8-20, 1726:4-12 (Sze Test.).) Dr. Sze has authored over 130 peer-reviewed publications, published chapters in most of the major textbooks in his field, and lectured and taught courses at universities and major radiology meetings, both nationally and internationally. (Trial Tr. at 1725:21–1726:3, 1726:13-17 (Sze Test.).) Imaging of pediatric patients makes up 50 percent of Dr. Sze's work at Yale, and 50 percent of the cases he has reviewed as an expert has been for plaintiffs. (Trial Tr. at 1727:10-23, 1759:12-14 (Sze Test.).)

31. Dr. Sze testified to the following opinions to a reasonable degree of medical probability at trial:

A. The initial imaging study available that Dr. Sze reviewed is the MR examination of A.H.'s brain from April 16, 2012, performed at CAMC. (Trial Tr. at 1729:18-22 (Sze Test.).) This study demonstrates enlarged ventricles due to brain tissue loss around those ventricles and mild thinning of the posterior body and splenium of the corpus callosum. (Trial Tr. at 1730:1-9, 1738:5-10 (Sze Test.).) As stated earlier, this is known as

periventricular leukomalacia, or "PVL" for short. (Trial Tr. at 1730:10-12 (Sze Test.).)

B. A follow-up MR examination of the brain was performed at Cincinnati Children's Hospital on March 3, 2016, and Dr. Sze reviewed it as well. (Trial Tr. at 1730:18-22 (Sze Test.).) This study demonstrated the same abnormal findings as the one from 2012, including PVL. (Trial Tr. at 1731:1-3 (Sze Test.).)

C. This series of imaging examinations demonstrates an appearance consistent with hypoxic-ischemic injury of the partial prolonged pattern in a premature stage of development. (Trial Tr. at 1738:25–1742:6, 1743:5-21, 1744:14-22 (Sze Test.).) This type of injury can be localized to the watershed regions or involve nearly all the cortex and subcortical white matter. (Trial Tr. at 1742:7-24 (Sze Test.).) In premature infants, the periventricular regions constitute the watershed regions and the abnormalities are typically periventricular in distribution. (Trial Tr. at 1742:20-24 (Sze Test.).)

D. In A.H.'s case, there is involvement of the periventricular regions, typical of the partial prolonged pattern of hypoxic-ischemic injury in a premature infant. (Trial Tr. at 1743:2-21 (Sze Test.).) A.H.'s scans showed misshapen ventricles with brightness immediately surrounding them, which is typical of PVL. (Trial Tr. at 1743:5-21 (Sze Test.).) The brightness, or injury, around the ventricles did not extend towards the periphery of the brain as is seen in term infants. (Trial Tr. at 1741:9-22, 1742:2-6, 1745:2-6 (Sze Test.).)

E. A.H. was born at 41 weeks of gestational age, (Trial Tr. at 1739:20-22 (Sze Test.)), but her imaging findings are typical of a premature pattern of injury, (Trial Tr. at 1743:2-3 (Sze Test.)). Thus, it is likely that the inciting events took place in utero, substantially prior to labor and delivery. (Trial Tr. at 1773:13-19 (Sze Test.).)

F. PVL is a hypoxic-ischemic injury that is "most common in infants of 32 gestational weeks or less." (Trial Tr. at 1743:22–1744:2 (Sze Test.).) PVL occurs during the development of the brain in utero at a premature stage of gestational fetal life, which is likely when A.H. suffered from an hypoxic-ischemic injury. (Trial Tr. at 1744:20-25, 1745:2-19 (Sze Test.).) While damage to white matter can occur in term infants, it is "much less likely when it's due to hypoxic-ischemic conditions." (Trial Tr. at 1760:12-19, 1761:3-7 (Sze Test.).) Plaintiffs' counsel attempted to discredit Dr. Sze on this point using medical literature that counsel disclosed for the first time at trial, but as Dr. Sze emphasized several times, those articles only bolstered the testimony he already provided—that it is much more likely for white matter injury in a periventricular distribution to occur in premature infants. (Trial Tr. at 1760:23–1771:5 (Sze Test.).)

G. Although A.H.'s injuries are consistent with hypoxic-ischemic events prior to 32 weeks of gestational age, the imaging appearance could also be due to genetic causes. (Trial Tr. at 1746:2-19 (Sze Test.).) For example, the MTHFR gene can affect the brain of developing infants by not transferring enough oxygenated blood through the mother's placenta, and this appeared

to have occurred in A.H.'s case most likely prior to 32 weeks of gestational age, (Trial Tr. at 1746:2-19 (Sze Test.)), because A.H. has an abnormality in her MTHFR gene, (ECF No. 286 (RGH Ex. 122)).[7] Plaintiffs' counsel's prolonged attempt to discredit Dr. Sze's testimony regarding the MTHFR gene based on the medical literature Dr. Sze relied upon was unsuccessful, at best. (Trial Tr. at 1747:15–1758:7 (Sze Test.).)

H. In sum, the radiographic evidence established that A.H.'s injury to her brain occurred in utero substantially prior to labor and delivery, many weeks prior to her birth at 41 weeks of gestation.[8]

32. The Court finds that the credibility of the United States' experts far outweighs that of Plaintiffs' experts regarding the issues in this case. Notably, several of Plaintiffs' main experts were acquired through expert referral services, such as Ellen Rieback's firm and the Expert Witness Institute, (Trial Tr. at 362:1–363:5 (Fassett Test.); Trial Tr. at 627:25–628:12 (O'Meara Test.); Trial Tr. at 428:5-20 (Connors Test.); Trial Tr. at 1067:8-11 (Rugino Test.)), whereas the United States' experts were retained based on their expertise in their particular subspecialities, (*see, e.g.*, Trial Tr. at 1812:8-9 (Scher Test.)).

[7] Dr. Jerome Barakos, Plaintiffs' radiology expert, excluded a genetic condition as a possible cause for A.H.'s condition based on a normal MR spectroscopy conducted in 2016. (Trial Tr. at 1176:24–1177:10 (Barakos Test.).) However, Dr. Barakos was unaware of A.H.'s MTHFR gene abnormality at the time of his deposition on April 2, 2021, despite the February 5, 2020 date on the result. (ECF No. 286 (RGH Ex. 122).)

[8] Plaintiffs' pediatric expert, Dr. O'Meara, testified as to the cause of A.H.'s neurologic injuries and relied on the 2012 and 2016 MRI reports in doing so. (Trial Tr. at 578:1–581:12 (O'Meara Test.).) However, Dr. O'Meara also admitted that she does not "have the skills and the ability to look at an MRI digitally and tell . . . what it shows" but rather "would need to rely on a pediatric neuroradiologist" for such an opinion. (Trial Tr. at 616:12-19 (O'Meara Test.).) Her opinion as to the timing of a hypoxic injury, which relies on what the 2012 and 2016 MRIs showed, contradicts with the opinion of Dr. Sze, Yale University School of Medicine's Chief of Neuroradiology for thirty years. Dr. Sze has much more credibility on this issue. Further, Dr. O'Meara did not review Dr. Sze's report at any point prior to testifying at trial. (Trial Tr. at 617:9-21 (O'Meara Test.).)

33. Regarding credibility, while Plaintiffs' midwife expert, Mr. Fassett, only practices midwifery in a clinical setting one day per week yet has been doing expert work for "[a]bout 25 years," (Trial Tr. at 262:22–263:3, 361:23-24 (Fassett Test.)), the United States' obstetrics expert, Dr. Landon, regularly delivers babies and sees patients in a clinical setting, (Trial Tr. at 1891:11-23 (Landon Test.)). Mr. Fassett has never published or conducted independent research within his field of practice, and he is not a member of the American College of Obstetricians and Gynecologists. (Trial Tr. at 359:24–360:4, 366:21-24 (Fassett Test.).) Dr. Landon has written over 200 peer-reviewed articles and over 80 book chapters in the fields of obstetrics and maternal-fetal medicine, and he belongs to a number of professional and scientific societies related to his fields of study, including the American College of Obstetrics and Gynecologists, the American Gynecological and Obstetrical Society, and the Society for Maternal-Fetal Medicine, among others. (Trial Tr. at 1895:12-17, 1893:24–1894:7 (Landon Test.).) Further, Mr. Fassett has never offered trial testimony on behalf of defendants, and this trial was the third he has been involved in with Plaintiffs' counsel's firm. (Trial Tr. at 359:5-12 (Fassett Test.).) Dr. Landon, on the other hand, has given most of his deposition testimony on behalf of plaintiffs and has offered trial testimony against defendants, including the United States, 50 percent of the time. (Trial Tr. at 1922:9–1923:7 (Landon Test.).)

34. Additionally, Plaintiffs' pediatric expert, Dr. O'Meara, is only trained and certified in general pediatrics and pediatric critical care (specializing in the life support of children)—she admitted that she has never published in the field of labor and delivery, does not go into delivery rooms as part of her specialty (and has not delivered a baby since she was in medical school, which she finished in 2000), has not worked in a newborn nursery since 2004, does not have familiarity with the interpretation of fetal monitoring strips, does not know a term-infant's oxygen saturation

level in utero, and is not a labor expert. (Trial Tr. at 551:17-18, 553:11-17, 563:7-21, 572:11-14, 604:15-23, 607:6-9, 610:17-20, 611:14-21, 655:5-24, 661:1-16, 662:2-8, 663:7-9 (O'Meara Test.). *Cf.* Trial Tr. at 1828:14-16 (Bedrick Test.) ("I care for critically ill babies in the newborn intensive care unit starting . . . ***in the delivery room until they are discharged from the nursery***." (emphasis added)).) Moreover, Dr. O'Meara could not name a single publication that she used to verify the opinions she gave in this case. (Trial Tr. at 615:5–616:4 (O'Meara Test.).) Additionally, Dr. O'Meara's testimony regarding A.H.'s Apgar scores, (Trial Tr. at 581:1-4 (testifying that seeing Apgar scores of seven and eight "immediately gets [her] attention that something happened" and means that it "was not a straightforward vaginal delivery")), not only is contrary to every other expert who testified in this case but also is contrary to all the medical and scientific literature referenced at trial. When confronted with a section from the 2014 Task Force Report that states "pretty unequivocally if the Apgar score at five minutes is greater than or equal to seven, it is unlikely that peripartum hypoxia-ischemia played a major role in causing neonatal encephalopathy," Dr. O'Meara's only response was: "The statement says that it's unlikely. It does not say that it rules it out." (Trial Tr. at 624:4-23 (O'Meara Test.).) Dr. O'Meara also was evasive at many times throughout her testimony, refusing to answer straightforward questions on cross-examination, (Trial Tr. at 639:24–645:11, 647:11-16, 649:17–650:4 (O'Meara Test.)), and her initial reaction to a question by Plaintiffs' counsel of whether she "[w]ould be willing . . . to submit transcripts of [her] deposition[] and [her] courtroom testimony for peer review in this case" was peculiar, at best, (Trial Tr. at 676:13-15 (O'Meara Test.) ("A. I have to clarify that. Is that – is that part of the routine peer review? I would – I mean, I'm happy for my peers to – to look at it, but – . . . .")). In giving her opinion on causation, Dr. O'Meara's testimony was based not on peer-reviewed publications that she could readily cite or widely accepted principles of medical or

scientific methodologies, but rather on the theory of Occam's razor as she believes that a hypoxic-ischemic event at or around the time of A.H.'s birth is "the simplest and most straightforward explanation" for A.H.'s neurologic abnormalities. (Trial Tr. at 679:22–680:6 (O'Meara Test.).) Given Dr. O'Meara's lack of experience with labor and delivery, diagnosing and treating neurological impairments, interpreting neuroradiological studies, or any reliance on the medical literature, her "simplest" explanation of the cause of A.H.'s abnormalities does not meet the standard for expert witness testimony required by Federal Rule of Evidence 702.

35. The Court also heard from various other experts who corroborated the opinions of the United States' witnesses:

A. Dr. Ernest Graham is board-certified in OB/GYN and maternal-fetal medicine, has worked as a Professor of OB/GYN at Johns Hopkins University for over twenty years (and for a number of years as the Director of the Combined Fellowship in Maternal-Fetal Medicine and Genetics), reviews articles for about twenty peer-reviewed journals, has authored seventeen books or book chapters, and has published 69 articles in peer-reviewed journals, of which many book chapters and articles address the subject of hypoxic-ischemic insults in the perinatal period in babies. (Trial Tr. at 918:20-23, 919:1-18, 920:12-24, 921:15-21, 925:1-7 (Graham Test.).) His unit covers labor and delivery at two hospitals on a day-to-day basis, year-round. (Trial Tr. at 919:19-24, 1089:21–1090:4 (Graham Test.).) Dr. Graham testified to a reasonable degree of medical probability that CNM Debra Crowder met the standard of care and that the fetal heart rate tracing showed no evidence of fetal distress—the tracing was "pretty ordinary."

(Trial Tr. at 927:7-21, 940:10-21 (Graham Test.) ("So I don't see any reason the midwife should be getting sued for medical malpractice in this case based on this heart rate tracing.").) He was asked several times throughout his review of the fetal monitoring strip at trial regarding what his response would be if CNM Crowder would have consulted him regarding the tracing, and every time he responded that he would have been reassured and would have advised to let Ms. Hysell keep laboring because there was no indication of a fetus in distress—"certainly not from lack of oxygen." (Trial Tr. at 935:14-21, 938:25–939:6 (Graham Test.).)

B. Dr. Peter Giannone, Chief of Neonatology at Kentucky Children's Hospital, has been practicing neonatology for almost twenty years since he finished his training as the Chief Fellow in the Neonatology Fellowship Program at Brown University. (Trial Tr. at 1517:11-12, 1518:23–1519:2 (Giannone Test.).) Dr. Giannone testified that there was not "a single feature in the accepted definition of neonatal encephalopathy that was present in this case." (Trial Tr. at 1555:19-23, 1559:8-19, 1561:3-11 (Giannone Test.).) He opined to a reasonable degree of medical probability that A.H. did not sustain brain damage "at any time from delivery through the time she was discharged" to days later and is not suffering today "as a result of untreated hypoxia during labor and delivery." (Trial Tr. at 1565:3-10 (Giannone Test.).) Dr. Giannone further testified, despite not giving an opinion as to the standard of care during labor and delivery, that "there was no indication during Ms. Hysell's course in labor and delivery that necessitated a

pediatrician, neonatologist or neonatal resuscitation team to be present at delivery." (Trial Tr. at 1582:19-25 (Giannone Test.).)

C. Dr. Andrea Gropman, Chief of Neurogenetics and Neurodevelopmental Pediatrics at George Washington University and Children's National Medical Center as well as Director of Neurodevelopmental Disability at Children's National, is board-certified in five areas (pediatrics, neurology with special competence in child neurology, medical genetics, neurodevelopmental disorders, and biochemical genetics), has written over 200 peer-reviewed publications, authored multiple chapters in books dealing with pediatric neurology and pediatric genetics, and serves as an editor on numerous peer-reviewed publications focused on issues present in this case. (Trial Tr. at 712:24–713:7, 718:5-19, 720:8-21, 721:21–722:14 (Gropman Test.).) Dr. Gropman opined to a reasonable degree of medical probability that A.H. did not experience brain-damaging hypoxia during the perinatal period or in the subsequent days during her stay in the nursery at RGH and that A.H. has an underlying genetic disorder that more likely than not caused her current neurological and developmental conditions. (Trial Tr. at 727:24–728:21, 730:13–731:15, 773:6–776:9 (Gropman Test.).) Dr. Gropman was unable to give a genetic diagnosis, however, because A.H. has not undergone whole exome sequencing to pinpoint which of A.H.'s 22,000 genes are defective. (Trial Tr. at 732:9-16, 734:21–735:7, 742:5–743:4 (Gropman Test.).) The capability to obtain whole exome sequencing and the ability to interpret its findings have improved and become more

accessible since A.H. was examined and tested by Dr. Schorry at Cincinnati Children's Hospital and even in the last two-and-a-half-years since Dr. Schorry's deposition (read into evidence at trial) was taken in 2019. (Trial Tr. at 731:18–732:16 (Gropman Test.).)

D. Dr. Gary Trock is board-certified in pediatrics, neurophysiology, neurology with special competence in child neurology, and sleep medicine, and he has practiced as a neurologist for approximately forty years. (Trial Tr. at 960:2–962:16 (Trock Test.).) Dr. Trock saw patients full time in his clinic until the COVID-19 global pandemic but still exams patients several days a week, many of whom have the diagnoses of cerebral palsy and ASD. (Trial Tr. at 962:23–963:22 (Trock Test.).) He was qualified at trial without objection "as an expert on the timing and potential cause of the developmental delays and other physical issues seen in [A.H.]" and "the cause of her autism and cerebral palsy," among other areas. (Trial Tr. at 966:8-15, 968:2-3.) Dr. Trock opined to a reasonable degree of medical probability that "what happened to [A.H.]'s brain [was] something that occurred well before 5:45 a.m. on October 29th, 2010, when Ms. Hysell presented to the hospital to begin labor and delivery." (Trial Tr. at 969:12-20, 970:5-9 (Trock Test.).) He further testified to a reasonable degree of medical probability that A.H. did not have neonatal encephalopathy, that her ASD was not caused by a hypoxic-ischemic insult, that A.H.'s MTHFR genetic mutation contributes to her current condition and her ASD more specifically, and that the PVL present in both of A.H.'s MRI scans "always

occurs, at the latest, 36 weeks – may begin as early as 28 weeks and up to 36 weeks. Beyond that it can't occur." (Trial Tr. at 972:19–973:5, 975:2-10, 979:4-18, 982:16–983:13, 996:9-13 (Trock Test.).) Finally, Dr. Trock opined to a reasonable degree of medical probability that CNM Debra Crowder did not "do things or fail to do things . . . that imposed a burden" on A.H. or her parents. (Trial Tr. at 996:4-8 (Trock Test.).)

E. Dr. Joshua Shimony, a board-certified physician in radiology (with a certificate in diagnostic radiology) and neuroradiology, has practiced as a neuroradiologist at Washington University Medical Center in St. Louis, Missouri, for twenty years and has served as the Director of Pediatric Neuroradiology at St. Louis Children's Hospital for the past ten years. (Trial Tr. at 1232:10-12, 1236:8-12, 1237:9-17 (Shimony Test.).) Dr. Shimony has published numerous peer-reviewed articles and book chapters in his field, has given lectures at events around the world, and has been recognized by the American Journal of Neuroradiology as one of the top reviewers in various peer-reviewed publications within his field of expertise. (Trial Tr. at 1239:10-19, 1243:1–1246:2 (Shimony Test.).) Dr. Shimony opined to a reasonable degree of medical probability that there was objectively verifiable evidence on A.H.'s brain imaging to show that her brain injury occurred not at or around the time of birth but rather "towards the end of the second trimester and the beginning of the third trimester of pregnancy." (Trial Tr. at 1256:11–1258:1, 1269:13-20, 1276:7-10, 1287:23–1288:13, 1326:4-17 (Shimony Test.).) The imaging patterns

that a pediatric neuroradiologist would expect to find if there was an event at or around the time of birth causing permanent brain damage to an infant, e.g., damage to the gray matter of the brain, were not present in A.H.'s MRI scans. (Trial Tr. at 1258:17–1262:23, 1283:14-25, 1318:4-12 (Shimony Test.). *See also* Trial Tr. at 1189:10–1190:3, 1191:23-25 (Barakos Test.).) Additionally, despite a prolonged attempt by Plaintiffs' counsel to get the witness to change his answer or contradict himself, Dr. Shimony consistently testified that to a reasonable degree of medical probability, it is more likely than not that the abnormality in A.H.'s MTHFR gene caused an in utero brain injury and that A.H. did not suffer from a perinatal hypoxic-ischemic insult. (Trial Tr. at 1307:19–1312:17 (Shimony Test.).)

36. The expert testimony and medical evidence presented by the United States at trial demonstrated, to a reasonable degree of medical probability, that Debra Crowder, CNM, did not breach the standard of care when rendering care to A.H. and her mother. Ms. Crowder always rendered health care services to Plaintiffs that complied with the standard of care applicable to her during the events involved in this case.

37. In addition, the expert testimony and medical evidence presented by the United States at trial established to a reasonable degree of medical probability that A.H.'s neurological problems were not proximately caused by hypoxia during labor and delivery. The most Plaintiffs could do at trial was suggest that hypoxia is one of a great number of possibilities that could have led to A.H.'s developmental delays. (*See, e.g.*, Trial Tr. at 1807:7-12 (Scher Test.) ("Q. You certainly do agree that all of the issues that [A.H.] has, all of the problems she has, are known to be caused by hypoxia; isn't that true? A. Hypothetically that's true, but not specifically for

[A.H.]'s history and specifics of her medical facts, no. But, yes, in general I say hypothetically yes."); Trial Tr. at 1944:18-22 (Landon Test.) ("Q. But, again, you have to agree that can also be caused by hypoxia, wouldn't you? A. I mean, any of these things could potentially, quote/unquote, be caused by hypoxia, but I'm asked to look at this in the totality of the circumstances.").) Thus, Plaintiffs' theory rests on pure possibility instead of medical probability. The electronic fetal monitoring strip pattern and Apgar scores were not consistent with hypoxia occurring during the labor process. As medical studies have shown, an Apgar score of seven or greater at five minutes with the type of electronic fetal monitoring tracing that existed in this case is not consistent with an acute hypoxic-ischemic event occurring at or near the time of birth as alleged by Plaintiffs in this case. In addition, A.H. was born with microcephaly, a clear indicator that her brain had not developed normally in utero. This finding also indicated that A.H. experienced a brain injury well prior to labor and delivery. The MRI scans also revealed that A.H. had PVL, which is an abnormality that occurs between 24 and 34 weeks of gestational age, and, thus, was not caused by labor and delivery, which occurred at 41 weeks of gestation. As explained by the expert witnesses of the United States, to a reasonable degree of medical probability, the medical evidence and A.H.'s clinical course at the time of labor and delivery were not indicative or consistent with a hypoxic-ischemic event occurring during labor and delivery.

38. Plaintiffs attempted to suggest the presence of a hypoxic-ischemic event through their midwife expert Mr. John Fassett's testimony about portions of the fetal monitoring strips that he claimed were "uninterpretable" and a period of time during labor where Ms. Hysell's oxygen saturation level measured 89 and 87, as well as through testimony of Mr. Hysell and Ms. Remines about the possibility of a cord issue during delivery.

A. Mr. Fassett had no criticisms of CNM Crowder "regarding any care that she gave from the time that she came on duty up through 12:22" on the day of delivery. (Trial Tr. at 349:7-10 (Fassett Test.).) His only criticisms came from the period of 12:22 to 14:19 despite agreeing that the fetal heart rate was detected at multiple points during that time and evidenced moderate variability and some accelerations. (Trial Tr. at 351:12–354:2, 360:19-23 (Fassett Test.).) With regard to the two-hour period where the mother's heart rate was being traced—notwithstanding the fact that the monitor also picked up A.H.'s heart rate in several places and showed good oxygenation—Mr. Fassett was only able to testify that "[***w***]***e don't know*** what happened for those two hours. There ***could have been*** – again, there ***could have been*** a prolonged bradycardia and an event – at that point a hypoxic event ***could have happened***. ***I don't know***. You don't know. I can just say, based on those two hours of an uninterpretable strip, ***I don't know*** what was going on with that baby during, you know, pushing." (Trial Tr. at 343:10-16 (Fassett Test.).) Mr. Fassett did not testify—let alone to a reasonable degree of medical probability—that any hypoxic event actually or even likely occurred. (Trial Tr. at 356:20-25 (Fassett Test.) ("Q. And so you can't testify to a reasonable degree of medical probability that during that period, from 1222 to 1419, when the strips were uninterpretable, you can't testify to a reasonable degree of medical probability that the baby was in fetal distress, can you? A. I cannot . . . ."), 360:24–361:2 (Fassett Test.).) This type of speculative testimony is excludable under Federal Rule of

Evidence 702. Further, while Mr. Fassett testified that an intrauterine pressure catheter ("IUPC") could have been placed to better track the fetal heart rate, (Trial Tr. at 330:13–331:16 (Fassett Test.)), he never testified that the failure to do so was a breach of the standard of care, and there is no evidence that the failure to place an IUPC proximately caused any injury to A.H.

B. Plaintiffs' counsel asked Mr. Fassett about the significance of Ms. Hysell having SaO_2 levels of 89 and 87 at one point early during labor, and Mr. Fassett began testifying that because "mom's not saturating at 95 percent, she's not sending good oxygen to the baby through the placenta system." (Trial Tr. at 292:21–293:11 (Fassett Test.).) Upon objection by the United States that Mr. Fassett had never expressed an opinion criticizing CNM Crowder about monitoring Ms. Hysell's SaO_2 levels, Plaintiffs' counsel stipulated that any testimony related to the mother's oxygen saturation levels was "not a criticism" but rather "an explanation of what the strip is showing." (Trial Tr. at 293:24–296:21.) Further, the SaO_2 measurements of 89 and 87 were taken at 08:30 and 08:36, respectively, (JM_1031)—during the time that Mr. Fassett testified he had no criticisms of CNM Crowder's care, (Trial Tr. at 349:7-10 (Fassett Test.)). During this time, Ms. Hysell was sitting for her epidural, which is not something midwives are involved in—it is handled by anesthesia personnel who also could have treated any problem related to Ms. Hysell's oxygen saturation rate while they were in the room. (Trial Tr. at 349:18–351:11 (Fassett Test.).)

Subsequently, Ms. Hysell had no more low oxygen saturation measurements.

C. Finally, Plaintiffs put forth testimony through Mr. Hysell and Ms. Remines that they heard CNM Debra Crowder mention the presence of a cord issue shortly before delivery. A prolapsed cord would be visible as it precedes the delivery of the baby. (Trial Tr. at 159:12-14 (Perkowski Test.).) Nurse Perkowski and CNM Crowder testified that there was no prolapsed cord issue in this case and there was no evidence of a nuchal cord or other cord problem. (Trial Tr. at 137:23-25, 152:1-6, 159:12-18 (Perkowksi Test.); Trial Tr. at 194:14-25, 195:1-8) (Crowder Test.).) Ms. Remines testified that the cord was not visible prior to delivery. (Trial Tr. at 1112:16-19, 1121:7-8, 1125:5-8 (Remines Test.).) In addition, the medical records do not indicate that a cord problem was encountered during delivery. (JM 1029–JM_1040, JM_1081, JM_2018.) Furthermore, Plaintiffs provided no expert testimony that any potential cord issue was handled inappropriately or that there was any negligent action or omission in response to such issue. In fact, Dr. Landon testified that CNM Crowder still met the standard of care even if this testimony was true, (Trial Tr. at 1915:16–1916:4 (Landon Test.)), and, as Dr. Landon testified, a cord compression coupled with a variable deceleration is not by itself an indication of hypoxia, (Trial Tr. at 1912:22-25 (Landon Test.)).

39. Plaintiffs failed to prove by a preponderance of the evidence that Debra Crowder, CNM, and the United States failed to exercise that degree of care, skill, and learning required or

expected of a reasonable, prudent health care provider in the profession or class to which she belonged, acting in the same or similar circumstances.

A. Plaintiff's midwife expert, Mr. Fassett, testified that midwives "take care of the normal to the low – to the low risk" patients and that if a midwife is "taking care of a patient who's higher risk, who's having fetal heart rate issues," it is the midwife's responsibility to call the obstetrician or "whoever [the] supervising physician is for that day, and to bring them into the room and to talk about th[e] strip." (Trial Tr. at 342:24–343:4 (Fassett Test.).) In this case, Ms. Hysell was not a high-risk patient, and there were no fetal heart rate issues that would warrant a call to a supervising physician. As the United States' expert in obstetrics, Dr. Landon, testified to a reasonable degree of medical probability, all CNM Crowder's actions and alleged inactions were within the applicable standard of care. (Trial Tr. at 1915:2-15, 1924:3-14 (Landon Test.).) Further, as maternal-fetal medicine expert Dr. Graham testified, if CNM Crowder would have consulted him regarding the tracing, he would have advised to let Ms. Hysell keep laboring because there was no indication of a fetus in distress. (Trial Tr. at 935:14-21, 938:25–939:6 (Graham Test.).)

B. The Court also finds that Plaintiffs' pediatric expert, Dr. O'Meara, was not offered as a standard of care expert as to midwives. (Trial Tr. at 566:14-28, 662:15-20.) She was not familiar with the standards of care applicable to nurse midwives at the time of the events involved in this case, had never supervised nurse midwives, never worked as a nurse midwife, and had never

trained nurse midwives. In addition, she was not familiar with the standards of care applicable to the interpretation of electronic fetal monitoring strips at the time of the events involved in this case. (Trial Tr. at 562:19-21 (O'Meara Test.).) Any testimony that she provided regarding the standard of care relating to resuscitative measures after birth are inapposite to CNM Crowder's role in this case as Plaintiffs' midwifery expert, Mr. Fassett, testified that he had no opinions or criticisms of the care rendered by CNM Crowder after the delivery of A.H. and agreed that once CNM Crowder handed A.H. to the nursing personnel after the delivery, her role with respect to A.H. ended. (Trial Tr. at 345:17-20, 348:10-25 (Fassett Test.).)

C. Plaintiffs' other experts—Dr. Rugino, Dr. Barakos, Ms. Connors, Ms. Lampton, and Mr. Staller—had no opinions as to midwifery standards of care or CNM Crowder's role in this case. (Trial Tr. at 406:19–407:5, 446:14-16 (Connors Test.); Trial Tr. at 662:15-20, 777:18–863:7 (Lampton Test.); Trial Tr. at 864:19–913:7 (Staller Test.); Trial Tr. at 1024:18–1076:25 (Rugino Test.).)

40. Plaintiffs also failed to prove by a preponderance of the evidence that any alleged negligence by Debra Crowder, CNM, and the United States proximately caused an injury to A.H. and Plaintiffs.

A. Dr. O'Meara's opinion that A.H. experienced hypoxia at or near the time of her birth was not supported by the medical evidence presented at trial. An Apgar score at five minutes that is equal to or greater than seven indicates that it is unlikely that peripartum hypoxia-ischemia played a major role in

causing a child's subsequently diagnosed neonatal encephalopathy. Plaintiffs' midwife expert also testified that the Apgar scores present in this case were normal. (Trial Tr. at 357:15-20, 360:17-18 (Fassett Test.).) In addition, the electronic fetal monitoring strip in this case was at least a Category II pattern based on the electronic fetal monitoring guidelines in effect at the time of A.H.'s birth. Notably, all fetal monitoring strips are at some point in time Category II, as up to 90% of all monitoring strips during labor and delivery will be in the broad Category II category. (Trial Tr. at 191:22-24 (Crowder Test.); Trial Tr. at 292:7-10 (Fassett Test.); Trial Tr. at 1901:22-25 (Landon Test.).) The fact that the strip remained at either a Category I or Category II the entire labor and delivery shows that no hypoxic event occurred during that time. (Trial Tr. at 192:7-8, 200:14–201:6 (Crowder Test.); Trial Tr. at 371:1-4 (Fassett Test.) ("A. . . . Once [the strip] gets to a Category III, you're pretty much done. Q. It can go from a III back to a II, correct? A. I've never seen that."); Trial Tr. at 1918:19-25 (Landon Test.).) Even Plaintiffs' midwife expert testified that the strip was never a Category III strip. (Trial Tr. at 357:9-10, 360:5-13 (Fassett Test.).) Further, the medical evidence indicated that a Category II pattern with an Apgar score of seven or greater at five minutes is not consistent with an acute hypoxic event. (Trial Tr. at 1919:1–1920:13 (Landon Test.) (discussing the 2014 Task Force Report).) Finally, A.H. did not experience any seizures, organ failure, or other signs of an acute hypoxic-ischemic event at the time of her birth or while she was in the nursery at RGH.

B. Neither Dr. Thomas Rugino nor Dr. Jerome Barakos, experts called by Plaintiffs, were able to testify to a reasonable degree of medical probability as to when A.H. experienced hypoxia. Regarding Dr. Rugino, Plaintiffs' pediatrics, physical medicine and rehabilitation, and neurodevelopmental disabilities expert, the parties filed a stipulation that is part of the record in this case that Dr. Rugino would not give causation opinions as to the timing of the injury other than it being sometime in the prenatal to perinatal period. (*See* ECF No. 267; *see also* Trial Tr. at 682:6–688:18, 1024:18–1076:25 (Rugino Test.) (failing to testify as to when the alleged hypoxic-ischemic injury occurred).) He did testify, however, that A.H. would have had autism regardless of any brain injury or anything that occurred during labor and delivery. (Trial Tr. at 1039:7-23, 1044:4-10 (Rugino Test.).) As for Dr. Barakos, Plaintiffs' radiology expert, when Plaintiffs' counsel discussed A.H.'s MRIs with him during his evidentiary deposition, the witness agreed with the 2016 MRI report finding that noted, "Periventricular white matter gliosis with mild to moderate volume loss, likely the sequelae of prior remote insult," (Trial Tr. at 1149:8-10 (Barakos Test.)), stating as follows: "This brain was damaged and this doctor is saying – is absolutely 100 percent accurate, it's because of an old injury. And – we stop there because – and we'll talk about this more. ***We can't tell when that injury occurred***, but we know it's due to an injury. And ***all we can say is prior – it's before – and it's remote***," (Trial Tr. at 1153:23–1154:3 (Barakos Test.)). Dr. Barakos did not provide an opinion to a reasonable degree of medical

probability that A.H. experienced an acute hypoxic-ischemic event at the time of her birth. (Trial Tr. at 1173:2-6 (Barakos Test.) ("This is a partial prolonged hypoxia that occurs, and we can say typically about – from about 26, 28 weeks gestation all the way through about a one to two-year-old. It's – it's in that spectrum that you get these characteristic imaging findings."), 1174:23-25 ("Q. All right. Is the – the periventricular leukomalacia from – from a perinatal hypoxic-ischemic injury? A. Yes. ***In the timeline I – the timeline*** [***that I put out***]. Q. Okay. Now, can an MRI tell us precisely when the injury occurred just from the MRI? Can you look at an MRI and say this occurred then or occurred then or occurred then? A. With the imaging findings we have here today, ***the answer is absolutely not, other than for the temporal resolution I described***."), 1180:1-3 ("So if you ask me did it happen at birth and when did it happen, that – I am deferring – that's going to be the job of the clinicians who care for the child . . . .").)

C. Plaintiffs' midwife expert, John Fassett, CNM, had no opinions on causation. (Trial Tr. at 358:7-18 (Fassett Test.).) The Court also finds that the opinions of Dr. Mark Landon, the obstetrical expert called by the United States, regarding the interpretation of the electronic fetal monitoring strips involved in this case were far more credible than those of Mr. Fassett.

D. A.H. had a head circumference at birth of 31.9 centimeters, which placed her head circumference at less than the 3rd percentile. (ECF No. 286-2 at 2 (RGH Ex. 149).) By definition, she was microcephalic because her head circumference was less than two standard deviations below the mean when

she was born. (Trial Tr. at 968:6-23 (Trock Test.); Trial Tr. at 1803:2-5 (Scher Test.); Trial Tr. at 1846:14–1847:4 (Bedrick Test.).) Even Plaintiffs' expert, Dr. Rugino, agreed that A.H. was microcephalic at birth because her head circumference was at the first percentile. (Trial Tr. at 1058:9-21 (Rugino Test.).) According to the medical evidence, microcephaly at birth may result from a prenatal insult early in the pregnancy or be the result of abnormal brain development in utero.

E. The MRI scans performed on A.H. in 2012 and 2016 revealed that she had PVL. PVL is caused by an event which usually occurs between 24 and 34 weeks of gestation. (Trial Tr. at 1326:4-14 (Shimony Test.) (reading statement from Brant and Helms' *Fundamental of Diagnostic Radiology* (5th ed.)).) This fact also indicates that A.H. experienced a brain injury long before she was born at 41 weeks of gestation.

F. The expert opinions on causation provided at trial by Dr. Mark Landon, Dr. Mark Scher, Dr. Alan Bedrick, and Dr. Gordon Sze, expert witnesses called by the United States, were far more credible than the causation opinions provided by Plaintiffs' expert witnesses, Dr. O'Meara, Dr. Rugino, and Dr. Barakos. The causation opinions of Drs. Landon, Scher, Bedrick, and Sze were fully supported by the medical evidence and medical literature while the causation opinions of Plaintiffs' experts, Drs. O'Meara, Barakos, and Rugino were not. None of Plaintiffs' experts testified to a reasonable degree of medical probability that CNM Crowder proximately caused an injury to Plaintiffs, including A.H.

G. Therefore, Plaintiffs not only failed to provide any credible evidence of a breach of the standard of care by Ms. Crowder and the United States, but they also failed to provide any credible evidence of a causal link between their alleged injuries and any alleged breaches of the standard of care by Ms. Crowder.

41. In summary, Plaintiffs failed to prove by a preponderance of the evidence, as required by W. Va. Code § 55-7B-3, that Debra Crowder, CNM, and the United States failed to exercise that degree of care, skill, and learning required or expected of a reasonable, prudent health care provider in the profession or class to which Ms. Crowder and the United States belonged, acting in the same or similar circumstances. Plaintiffs also failed to prove by a preponderance of the evidence that any negligence by Debra Crowder, CNM, and the United States proximately caused an injury to the plaintiffs.

II. PROPOSED CONCLUSIONS OF LAW

1. Jurisdiction of this action is predicated on the Federal Tort Claims Act ("FTCA"), 28 U.S.C. §§ 1346(b), 2401(b) and 2671, *et seq.*

2. Venue is proper under 28 U.S.C. § 1402(b).

3. Plaintiffs submitted an administrative tort claim on January 29, 2018, which was administratively denied on May 29, 2018, by the United States Department of Health and Human Services.

4. Access Health and Debra Crowder, CNM, were deemed to be employees of the United States under the Public Health Service Act and actions against them are treated as actions against the United States. *See* Federally Supported Health Centers Assistance Act of 1992, Pub. L. No. 102-501, 106 Stat. 3268 (codified as amended at 42 U.S.C. § 233(g)-(n) (1998)).

5. The FTCA provides a judicial remedy to those who suffer injury or damage as a result of the negligence of employees of the federal agencies of the United States Government. It permits recovery on claims for money damages "for . . . personal injury or death caused by the negligent or wrongful act or omission of any employee of the Government while acting within the scope of his office or employment, under circumstances where the United States, if a private person, would be liable to the claimant in accordance with the law of the place where the act or omission occurred." 28 U.S.C. § 1346(b). *See also* 28 U.S.C. § 2674; *Bellomy v. United States*, 888 F. Supp. 760 (S.D. W. Va. 1995). The substantive elements of a negligence action brought under the FTCA are generally governed by the law of the state in which the negligence occurred. *United States v. Muniz*, 374 U.S. 150 (1963); *Doganieri v. United States*, 520 F. Supp. 1093, 1095 (N.D. W. Va. 1981).

6. The Fourth Circuit and this Court have long recognized that the Medical Professional Liability Act ("MPLA"), W. Va. Code § 55-7B-1, *et seq.*, applies in FTCA cases alleging medical negligence occurring in West Virginia. Because Plaintiffs allege that the purported negligence occurred in West Virginia, the Court is bound to apply West Virginia's substantive law, which, in cases such as this one involving medical negligence, is the MPLA. *See Butts v. United States*, 930 F.3d 234, 238–39 (4th Cir. 2019), *cert. denied*, 140 S. Ct. 1113 (2020); *Pearson v. Panaguiton*, 699 F. App'x 174 (4th Cir. 2017); *Simms v. United States*, 839 F.3d 364, 370–73 (4th Cir. 2016); *Drennen v. United States*, 375 F. App'x 299 (4th Cir. 2010); *Osborne v. United States*, 166 F. Supp. 2d 479 (S.D. W. Va. 2001); *Bellomy*, 888 F. Supp. at 760.

7. Under West Virginia law, negligence actions involving health care services rendered by a health care provider such as Ms. Crowder are governed by the MPLA. The burden

of proof imposed upon plaintiffs seeking to recover damages for alleged medical negligence is set forth under the MPLA, which states:

> § 55-7B-3. Elements of proof.
>
> (a) The following are necessary elements of proof that an injury or death resulted from the failure of a health care provider to follow the accepted standard of care:
>
> > (1) The health care provider failed to exercise that degree of care, skill and learning required or expected of a reasonable, prudent health care provider in the profession or class to which the health care provider belongs acting in the same or similar circumstances; and
> >
> > (2) Such failure was a proximate cause of the injury or death . . . .

W. Va. Code § 55-7B-3. *See also Butts*, 930 F.3d at 238–39; *Rubin v. United States*, 88 F. Supp. 2d 581, 597 (S.D. W. Va. 1999); *MacDonald v. City Hosp., Inc.*, 2715 S.E.2d 405, 423 n.22 (W. Va. 2011). Other aspects of this action are governed by the common law of West Virginia. *See Bellomy*, 888 F. Supp. at 763–64.

8. To prevail on a claim under the MPLA, the burden is on the plaintiff to prove, by a preponderance of the evidence, that the defendant was negligent (breached the applicable standard of care) and that the negligence (breach of the applicable standard of care) was a proximate cause of the plaintiff's injury. *See Butts*, 930 F.3d at 238–39; *Sexton v. Greico*, 613 S.E.2d 81, 83 (W. Va. 2005) (per curiam) (quoting Syl. pt. 2, *Walton v. Given*, 215 S.E.2d 647 (W. Va. 1975)).

9. Under W. Va. Code § 55-7B-3, plaintiffs have the burden of proving both a breach of the standard of care and that any alleged breach proximately caused the plaintiffs' alleged injuries and damages. To establish breach of the standard of care, West Virginia law requires a party bringing a medical malpractice claim to show that "[t]he health care provider failed to exercise that degree of care, skill and learning required or expected of a reasonable, prudent health

care provider in the profession or class to which the health care provider belongs acting in the same or similar circumstances . . . ." W. Va. Code § 55-7B-3(a)(1).

10. The applicable standard of care, and the defendant's failure to meet the standard of care, must be established by the "testimony of one or more knowledgeable, competent expert witnesses if required by the court." *Id.* § 55-7B-7; *Butts*, 930 F.3d at 238–39. *See also MacDonald*, 715 S.E.2d at 423 n.22. "It is the general rule that in medical malpractice cases negligence or want of professional skill can be proved only by expert witnesses." Syl. Pt. 2, *Roberts v. Gale*, 139 S.E.2d 272 (W. Va. 1964), *quoted in* Syl. pt. 1, *Neary v. Charleston Area Med. Ctr.*, 460 S.E.2d 464 (W. Va. 1995) (per curiam); Syl. pt. 1, *Farley v. Meadow*s, 404 S.E.2d 537 (W. Va. 1991).

11. The MPLA discusses the requisites for expert witness testimony on the standard of care:

> § 55-7B-7. Testimony of expert witness on standard of care.
>
> (a) The applicable standard of care and a defendant's failure to meet the standard of care, if at issue, shall be established in medical professional liability cases by the plaintiff by testimony of one or more knowledgeable, competent expert witnesses if required by the court. A proposed expert witness may only be found competent to testify if the foundation for his or her testimony is first laid establishing that: (1) The opinion is actually held by the expert witness; (2) the opinion can be testified to with reasonable medical probability; (3) the expert witness possesses professional knowledge and expertise coupled with knowledge of the applicable standard of care to which his or her expert opinion testimony is addressed; (4) the expert witness's opinion is grounded on scientifically valid peer-reviewed studies if available; (5) the expert witness maintains a current license to practice medicine with the appropriate licensing authority of any state of the United States: Provided, That the expert witness's license has not been revoked or suspended in the past year in any state; and (6) the expert witness is engaged or qualified in a medical field in which the practitioner has experience and/or training in diagnosing or treating injuries or conditions similar to those of the patient. If the witness meets all of these qualifications and devoted, at the time of the medical injury, sixty percent of his or her professional time annually to the active clinical practice in his or her medical field or specialty, or to teaching in his or her medical field or specialty in an accredited university, there shall be a rebuttable presumption that the witness is qualified as an expert. The parties shall have the opportunity to impeach any witness's qualifications as an expert. Financial records of an expert witness are not

> discoverable or relevant to prove the amount of time the expert witness spends in active practice or teaching in his or her medical field unless good cause can be shown to the court.
>
> (b) Nothing contained in this section limits a trial court's discretion to determine the competency or lack of competency of a witness on a ground not specifically enumerated in this section.

W. Va. Code § 55-7B-7.

12. "[C]asual familiarity with the standard of care and treatment commonly practiced by physicians engaged in the defendant's specialty" is not enough to offer expert testimony on the standard of care. *Gilman v. Choi*, 406 S.E.2d 200, 204 (W. Va. 1990), *overruled on other grounds*, *Mayhorn v. Logan Med. Found.*, 454 S.E.2d 87 (W. Va. 1994). A medical expert's ability to testify as to a particular standard of care may be acquired "through practical experience, recent formal training and study or a combination of these factors." *Id.* "While there is no dearth of case law to show that under certain circumstances, a physician from one specialty may opine as to the breach of the standard of care of a physician from another specialty, where a physician from one specialty cannot demonstrate the requisite training, experience, or familiarity with the standard of care applicable to that of the defendant physician, such testimony is barred." *Brown v. U.S. Dep't of Justice*, No. 1:17cv144, 2019 WL 2488760, at *26 (N.D. W. Va. Jan. 23, 2019) (footnotes omitted), *report and recommendation adopted*, 2019 WL 1234324, at *1 (N.D. W. Va. Mar. 18, 2019), *aff'd*, 787 F. App'x 155 (4th Cir. 2019). *See also Farley v. Shook*, 629 S.E.2d 739, 745 (W. Va. 2006) (barring standard of care testimony from emergency room physician).

13. "Questions of an expert's credibility and the weight accorded to his testimony are ultimately for the trier of fact to determine." *Arkwright Mut. Ins. Co. v. Gwinner Oil, Inc.*, 125 F.3d 1176, 1183 (8th Cir. 1997). However, the Court is not required to accept as true and may disregard expert testimony that is internally inconsistent or contradictory to the facts proven at

trial. *Holm v. United States*, 325 F.2d 44, 46 (9th Cir. 1963); *Jones v. Heckler*, 614 F. Supp. 277, 280 (D. Vt. 1985) (disregarding medical expert's testimony as not probative of plaintiff's medical condition because testimony was internally inconsistent and contradicted by other medical evidence of record).

14. As the Supreme Court and the Fourth Circuit have held, expert testimony that is speculative is inadmissible, lacks probative value, and should be excluded. Moreover, an expert's opinion is inadmissible when it is based on assumptions that are speculative and are not supported by the record. *See Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579, 589–91 (1993) (speculative expert testimony is not admissible); *Tyger Const. Co. Inc. v. Pensacola Const. Co.*, 29 F.3d 137, 142 (4th Cir. 1994) (expert testimony that is based on speculation is not admissible under Fed. R. Evid. 702); *Vincent v. United Techs. Corp.*, No. 88–1006, 1988 WL 83389, at *1 (4th Cir. July 29, 1988) (expert testimony that is speculation properly excluded as being inadmissible and lacking in probative value). If an expert witness cannot testify to a reasonable degree of probability or certainty regarding an opinion, then that opinion is inadmissible and should be excluded. *See Waffen v. U.S. Dep't of Health & Human Servs.*, 799 F.2d 911, 918 (4th Cir. 1986) ("The mere possibility that a defendant's conduct may have caused injury does not provide sufficient causation as a basis for liability. The plaintiff has the burden of introducing evidence which affords a reasonable basis for the conclusion that it is more likely than not that the conduct of the defendant was a substantial factor in bringing about the result. When the matter remains one of pure speculation or conjecture, the court must direct a verdict for the defendant."); *Spain v. Powell*, 90 F.2d 580, 582 (4th Cir. 1937) ("We have then an uncertainty as to the inferences which may fairly be drawn from the evidence, and the judgment as a matter of law must go against the party upon whom rests the necessity of showing that he is entitled to recover.");

Huskey v. Ethicon, *Inc.*, 29 F. Supp. 3d 691, 729 (S.D. W. Va. 2014) (finding that the expert could not say one way or the other whether prior back surgery was related to patient's pelvic pain complaints and holding the testimony of the expert inadmissible, stating that "[n]ot only is her opinion speculation, but it is not helpful to the jury because it is not made to a reasonable degree of medical certainty").

15. While the MPLA has changed many of the common law principles of medical negligence actions, it has not totally displaced the common law for medical negligence actions. These common law principles amplify and explain many of the concepts and provisions of the MPLA. Under the common law, it is the duty of a health care provider to use reasonable and ordinary care in the diagnosis and treatment of a patient. *Utter v. United Hosp. Ctr., Inc.*, 236 S.E.2d 213 (W. Va. 1977). A physician is not required to provide a patient with "the highest degree of care possible." *Bellomy*, 888 F. Supp. at 765 (citing *Schroeder v. Adkins*, 141 S.E.2d 352, 357 (W. Va. 1965)). "Moreover, where there is more than one method of medical treatment accepted and applied by average physicians similarly situated, the physician may take into account the particular circumstances of each case and may exercise his honest and best judgment in selecting a course of treatment for individual patients." *Id.* at 765–66. In fact, if there is more than one acceptable method of treatment, the physician need not choose the best one. *Id.* at 766 (citing *Maxwell v. Howell*, 174 S.E. 553, 554–55 (W. Va. 1934)). *See also Butts*, 930 F.3d at 238–39.

16. Whether a health care provider breached the applicable standard of care is to be judged at the time of his or her alleged negligent acts. *Bellomy*, 888 F. Supp. at 765 (citing Syl. pt. 2, *Schroeder*, 141 S.E.2d at 352).

17. While a plaintiff has no duty to exclude every other plausible theory as to the cause and effect of his injury, the plaintiff must establish a theory of causation by a preponderance of the

evidence. *Long v. City of Weirton*, 214 S.E.2d 832, 846–48 (W. Va. 1975). However, proof that a medical care provider failed to cure an affliction or effect a perfect remedy for an injury does not alone establish negligence or lack of skill. A bad result does not necessarily mean that the medical care provider deviated from the standard of care. *Schroeder*, 141 S.E.2d at 357–58.

18. To be actionable, negligence must be a proximate cause of the injury complained of and must be such as might have been reasonably expected to produce an injury. *See* W. Va. Code § 55-7B-3; *Mays v. Chang*, 579 S.E.2d 561, 566 (W. Va. 2003). A proximate cause is that cause which, if unbroken by any efficient intervening cause, produces the injury and without which there would not have been the same result. *Mays*, 579 S.E.2d at 566. "In a malpractice case, the plaintiff must not only prove negligence but must also show that such negligence was the proximate cause of the injury." Syl. Pt. 4, *Short v. Appalachian OH–9, Inc*., 507 S.E.2d 124 (W. Va. 1998). *See also Dellinger v. Pediatrix Med. Grp.*, 750 S.E.2d 668, 676 (W. Va. 2013) (citing *Short*, 507 S.E.2d at 124) (plaintiff must prove both negligence and proximate causation).

19. To establish proximate causation in a medical negligence case, a plaintiff must prove such a causal link through expert witness testimony. *See Fitzgerald v. Manning*, 679 F.2d 341, 350 (4th Cir. 1982) ("Just as negligence or violation of the standard of care must ordinarily rest on expert opinion evidence, so proof of causation—that is that the defendant's negligence was 'more likely' or 'more probably' the cause of the plaintiff's injury—requires expert testimony." (footnote omitted)); *Bellomy*, 888 F. Supp. at 764 (malpractice case and causation must ordinarily be proven through expert testimony). The failure to prove the causal link by expert witness testimony between the alleged negligence and the alleged injury means that the plaintiff has a fatal defect in his or her case which requires dismissal of the case. *See Nottingham v. United States*, No. 2:16-cv-03022, 2017 WL 3026926, at *7 (S.D. W. Va. July 17, 2017) (dismissing medical

malpractice case because the plaintiff failed to provide proper expert testimony under W. Va. Code § 55-7B-3 to establish a breach in the standard of care and that the purported breach proximately caused the alleged injury); *Dellinger*, 750 S.E.2d at 677 ("The lack of expert medical testimony as to causation was therefore equally fatal to petitioner's case as her failure to present a disputed issue of material fact on medical negligence."); *Farley*, 629 S.E.2d at 745 ("Thus, because Dr. Weihl was the only expert designated to provide standard of care and causation testimony against the emergency room physician and the hospital, and because he was unable to provide the necessary causal links, the Farleys were unable to prove their case against these two appellees. The circuit court was correct in awarding summary judgment to Dr. Fornari and St. Mary's, and we accordingly affirm the circuit court's ruling."); *Short*, 507 S.E.2d at 131–32 (failure to present expert witness testimony on causation was fatal to plaintiff's case and required entry of summary judgment); *Hicks v. Chevy*, 358 S.E.2d 303, 305 (W. Va. 1987) ("Proof that the negligence or want of professional skill was the proximate cause of the injury of which the plaintiff complains must ordinarily be by expert testimony as well.").

20. Even if an injury has occurred, if that injury is not linked to a breach in the standard of care by the plaintiff through expert witness testimony, then the burden of proof under W. Va. Code § 55-7B-3 is not met. *See Fitzgerald*, 679 F.2d at 350; *Bellomy*, 888 F. Supp. at 764; *Dellinger*, 750 S.E.2d at 676; *Farley*, 629 S.E.2d at 745; *Short*, 507 S.E.2d at 131–32; *Hicks*, 358 S.E.2d at 305. In other words, in the absence of expert testimony causally connecting A.H.'s injuries to CNM Debra Crowder's alleged negligence, proximate cause cannot otherwise be inferred because such an inference would be wholly based on "abject speculation." *See Dellinger*, 750 S.E.2d at 677 & n.15 (explaining the limited use of the "reasonable inference" theory in establishing causation in medical malpractice trials). The element can only be met if expert

testimony has established that the alleged negligence was "more likely" than not the cause of the plaintiff's injury. *See Fitzgerald*, 679 F.2d at 350; *Waffen*, 799 F.2d at 918.

21. If a plaintiff cannot establish an essential element of his or her case, all other facts become immaterial, and the court has an affirmative obligation to prevent the case from going forward. *Drewitt v. Pratt*, 999 F.2d 774, 778 (4th Cir. 1993) (applying rule in summary judgment context).

22. While expert testimony on the issue of causation need only be stated to a reasonable degree of probability, proof of any future damages must be established to a reasonable degree of certainty. *See Adkins v. Foster*, 421 S.E.2d 271 (W. Va. 1992); *Jordan v. Bero*, 210 S.E.2d 618 (W. Va. 1974). In computing any award for a plaintiff's lost future earnings and fringe benefits, the award must be discounted or reduced for the increasing likelihood that she would not have remained in employment so long. *Flannery v. United States*, 718 F.2d 108 (4th Cir. 1983), *cert. denied*, 467 U.S. 1226 (1984). Then, that amount must be reduced to present value because a sum received today can be invested and earn money at current interest rates. *Gault v. Monongahela Power Co.*, 223 S.E.2d 421 (W. Va. 1976); *Salerno v. Manchin*, 213 S.E.2d 805 (W. Va. 1974).

23. Limits were also imposed on non-economic losses of $250,000 unless those losses were for wrongful death, permanent and substantial physical deformity, loss of use of a limb or a bodily organ system, or a permanent physical or mental functional injury that permanently prevents the injured person from being able to independently care for him or herself and perform life sustaining activities, in which case the damages are capped at $500,000. W. Va. Code § 55-7B-8. Beginning in January 2004, the $250,000 and $500,000 caps began rising by the consumer price index up to fifty percent of the capped amounts. In the event that the caps on damages are found to be invalid for any reason, then the savings clause caps the damages at one million dollars.

W. Va. Code § 55-7B-8(c). The caps adopted in 2003 which became effective in 2004 have since been found to be valid and constitutional. *McDonald v. City Hosp., Inc.*, 715 S.E.2d 405 (W. Va. 2011).

24. Plaintiffs have the burden to prove that portion of the damages which is attributable to the alleged negligence of the United States. The United States cannot be held liable for the damages and expenses (such as, but not limited to, medical expenses, lost earning capacity or lost wages, pain and suffering, and other alleged damages) which Plaintiffs would have experienced for the treatment and problems attributable to any underlying disease or medical condition not proximately caused by the alleged negligence of the United States. If Plaintiffs cannot meet their burden of proof on this issue, then any medical expenses, lost earning capacity or lost wages, expenses, or other damages attributable to the underlying disease or medical condition not proximately caused by the alleged negligence must be excluded. *Abdulla v. Pittsburgh & Weirton Bus Co.*, 213 S.E.2d 810 (W. Va. 1975).

25. The plaintiffs have no right to a jury trial under the FTCA. *See* 28 U.S.C. § 2402. *See also Lehman v. Nakshian*, 453 U.S. 156, 161 (1981) ("Finally, in tort actions against the United States, *see* 28 U.S.C. § 1346(b), Congress has similarly provided that trials shall be to the court without a jury. 28 U.S.C. § 2402." (footnote omitted)); *Carlson v. Green*, 446 U.S. 14, 22 (1980) ("Third, a plaintiff cannot opt for a jury in an FTCA action, 28 U.S.C. § 2402."); *Gutierrez De Martinez v. D.E.A.*, 111 F.3d 1148, 1152 (4th Cir. 1997) ("[T]he denial of a jury trial under the FTCA, does not offend the Seventh Amendment."). While Federal Rule of Civil Procedure 39(c) allows the Court discretion to use an advisory jury in actions not otherwise triable by a jury, the Court is in no way bound by the advisory verdict. *See, e.g.*, *Kaniff v. United States*, 351 F.3d 780,

781 (7th Cir. 2003) (affirming district court's decision to disagree with the recommendation of the advisory jury in FTCA case and enter judgment for the United States).

III. CONCLUSION

1. Plaintiffs failed to meet their burden under W. Va. Code § 55-7B-3. Plaintiffs failed to prove by a preponderance of the evidence that Debra Crowder did not exercise that degree of care, skill and learning required or expected of a reasonable, prudent health care provider in the profession or class to which the health care provider belongs acting in the same or similar circumstances.

2. Plaintiffs also failed to prove by a preponderance of the evidence that any alleged negligent act or omission by Debra Crowder proximately caused the alleged injuries to A.H. and Plaintiffs.

3. Therefore, the Court finds that the United States is not liable to Plaintiffs.

4. The Court enters Judgment in favor of the United States and against Plaintiffs.

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IN THE UNITED STATES DISTRICT COURT
FOR THE SOUTHERN DISTRICT OF WEST VIRGINIA
AT BECKLEY

RYAN HYSELL and CRYSTAL HYSELL,
on behalf of their daughter, A.H., a minor,

Plaintiffs,

v. Civil Action No. 5:18-cv-01375

RALEIGH GENERAL HOSPITAL,
and THE UNITED STATES OF AMERICA,

Defendants.

CERTIFICATE OF SERVICE

I hereby certify that on July 15, 2021, I electronically filed the foregoing **PROPOSED FINDINGS OF FACT AND CONCLUSIONS OF LAW OF THE UNITED STATES OF AMERICA** with the Clerk of the Court using the CM/ECF system which will send notification to the following CM/ECF participants:

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